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B R A I N :
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VOL. VIII.



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A JOURNAL OF NEUROLOGY.

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CONTENTS.

ORIGINAL ARTICLES:—

	PAGE
Cases of Recovery from Symptoms pointing to the presence of Organic Cerebral Disease. By J. S. Bristowe, M.D., F.R.S.	1
Nature of the Spinal Lesion in Polio-myelitis Anterior Acuta, or Infantile Paralysis. By D. Drummond, M.A., M.D.	14
On some Central Affections of Vision. By W. J. Dodds, M.D., D.Sc. 21,	345
An Analysis of the Nerve-Phenomena in a Case of Anaesthetic Leprosy. By W. Allen Sturge, M.D.	40
General Paralysis of the Insane. A Study of the Deep Reflexes, etc., of the Spinal Cord. By W. C. Beatley, M.D.	65
On the Corpus Callosum in the Embryo. By Prof. Hamilton, M.B., F.R.S.E.	145
On some of the Rarer Forms of Muscular Atrophies. By Prof. Dreschfeld, M.D., F.R.C.P.	164
An Experimental Enquiry into the Nature of the Objective Cause of Sensation. By Prof. Haycraft, M.B., B.Sc., F.R.S.E.	191
Sensation and Movement. By Ch. Féré, M.D.	210
Nerve Troubles as Foreshadowed in the Child. By Ch. Féré, M.D.	230
On Staining "in Toto" the Central Nervous System with Weigert's Hematoxylin. By Charles E. Beevor, M.D., M.R.C.P.	239
On a Muscular Phenomenon observed in Hysteria, and analogous to the "Paradoxical Contraction." By Prof. Charcot and Dr. Richer	289
The Inertia of the Eye and Brain. By James M'Keen Cattell	295
Cases of Ophthalmoplegia, complicated with various other Affections of the Nervous System. By John S. Bristowe, M.D., LL.D., F.R.S.	313
A Note on Spastic Paraplegia and the Treatment of some Cases by Rest. By H. B. Donkin, M.B. (Oxon.), F.R.C.P.	370
On Professor Hamilton's Theory concerning the Corpus Callosum. By C. Beevor, M.D., M.R.C.P.	377
On a New Induction Apparatus. By Prof. E. Tiegel, M.D.	380
Further Observations on Alcoholic Paralysis. By Prof. J. Dreschfeld, M.D., F.R.C.P.	433
On a Case of Multiple Spinal and Cerebral Tumours (Sarcomata), etc. By Dr. Thomas Harris, M.D. (Lond.)	447
On a Case Illustrating the Cortical Nature of Epilepsy, etc. By D. Noël-Paton, M.D., B.Sc.	474
On a Case of Amnesia. By E. A. Dingley, M.D. (Lond.)	492
On a Case of Bilateral Degeneration in the Spinal Cord, etc. By W. B. Hadden, M.D., and C. S. Sherrington, M.B.	502
The Influence of the Intensity of the Stimulus on the Length of the Reaction Time. By James M'Keen Cattell	512

CLINICAL CASES:—

	PAGE
Case of Brachial Monoplegia, due to Lesion of the Internal Capsule. By A. H. Bennett, M.D., and C. M. Campbell, M.D.	78
An Unusual Case of Athetosis. By Seymour J. Sharkey, M.B.	85
Case of Perforating Tuberculosis of Skull with Cerebral Symptoms. By Walter Edmunds, F.R.C.S.	88
Case of Pachymeningitis Cervicalis Hypertrophica. By C. W. Suckling, M.D. (Lond.), M.R.C.P.	91
Two Cases of Spinal Disease associated with Insanity:—I. Tabes Dor- salis; and II. Chronic Atrophic Spinal Paralysis. By William Dudley, M.B. (Lond.)	243
Case of Cerebral Abscess. By Arthur E. W. Fox, M.B., F.R.C.P. (Edin.)	251
Nerve Suture; Strangulation at Point of Junction; Operation. Rapid Recovery of Sensation and Motion. By Walter Pye, F.R.C.S.	255
A Case of Syphilitic Disease of Cerebral Arteries. By Edward O. Daly, M.A., M.D. (Oxon.), M.R.C.P.	392
A Case of Hystero-Epilepsy in the Male. By James Oliver, M.B. (Edin.), M.R.C.P. (Lond.)	397
Two Fatal Cases of Tetanus. By W. B. Hadden, M.D.	401
Case of almost complete Destruction of the Right Hemisphere of the Cerebellum, &c. By George Ogilvie, B.Sc., M.B.	405
Notes and Remarks upon a Case of Villous Tumour in the Fourth Ventricle. By J. Harrington Douty	409
Case of Calcareous Gumma in the Brain. By G. Lacy Barritt, M.R.C.S., L.R.C.P. (Lond.)	413
Case of Amyotrophic Lateral Sclerosis. By C. E. Becvor, M.D. (With a Note on the Jaw-Jerk by Dr. de Watteville)	516
Primary Spastic Paralysis and Pseudo-Hypertrophic Paralysis in Dif- ferent Members of a Family. By R. W. Philip, M.A.	520
Hystero-Catalepsy in a Male; Attacks suspended by Testicular Pressure. By A. McLane-Hamilton, M.D. (New York)	528
A Case of Multiple Simultaneous Cerebral Hemorrhages, &c. By W. Hale White, M.D.	532

CRITICAL DIGEST:—On the Combination of Lateral and Posterior Sclerosis in the Spinal Cord. By J. A. Ormerod, M.D.	110
--	-----

REPORT:—On Recent Advances in the Anatomy of the Nervous System. By James Anderson, M.D.	125
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REVIEWS AND NOTICES OF BOOKS:—

Sully: Outlines of Psychology—Morell: An Introduction to Mental Philosophy on the Inductive Method. By Charles Mercier.	94
Althaus on Sclerosis of Spinal Cord, &c. By Prof. Schultze	96

Schäfer and Horsley on the Functions of the Marginal Convolution— Levillain : Histoire et Critique des progrès réalisés dans l'étude des fonctions du cerveau—Luys on the Brain and its Functions— Bastian on the Brain as an Organ of Mind—Kusssmaul : Les Troubles de la Parole—Morselli : Rivista di Filosofia Scientifica— Sergi : L'Origine dei fenomeni psichici—Müller : Zur Einleitung in die Elektrotherapie—Smith : A Practical Treatise on Disease in Children. By A. de Watteville	98
Sankey : Lectures on Mental Disease. By J. S. Bristowe, M.D.	108
Stewart : An Introduction to the Study of the Diseases of the Nervous System—Bramwell on Diseases of the Spinal Cord. By H. B. Donkin, M.D.	109
Ribot : Les Maladies de la Personnalité. By Charles Mercier	259
Mauthner : Gehirn und Auge—Wilbrand : Ophthalmiatriische Beiträge zur Diagnostik der Gehirnkrankheiten. By James Anderson, M.D.	263
Bernard : De l'Aphasie et de ses différentes Formes—Dalton : The Topographical Anatomy of the Brain—Fage : Injuries of the Spine and Spinal Cord without apparent Mechanical Lesion, and Nervous Shock in their Surgical and Medico-Legal Aspects. By A. de Watteville	266
Foville : La Législation relative aux Aliénés en Angleterre et en Ecosse. By John Charles Bucknill	415
Richer : Etudes Cliniques sur la Grande Hystérie, ou Hystéro-épilepsie. By A. de Watteville	422
Ross : A Treatise on the Diseases of the Nervous System. By J. Hughlings-Jackson	423
Gowers : Lectures on the Diagnosis of Diseases of the Brain. By T. Buzzard	425
Landois : Text-book of Human Physiology. By A. Waller, M.D.	535
Marique : Recherches expérimentales sur la mécanique de fonctionne- ment des Centres Psychomoteurs du Cerveau. By A. de Watte- ville	536
Edinger : Zehn Vorlesungen über den Bau der Nervösen Centralorgane —Heiberg : Scheme of the Functions of the Cerebral Nerves— Report on Recent Advances in the Anatomy of the Nervous System. By James Anderson, M.D.	539

ABSTRACTS OF BRITISH AND FOREIGN JOURNALS :—

Horsley on Substitution—Sharkey and Lawford on Acute Optic Neuritis —Sharkey on Embolism of the right Middle Cerebral Artery, &c., and on Homonymous Hemianopia. By A. Hughes Bennett.	132
Marie on Disseminated Sclerosis. By H. H. Tooth	137
Burnett and Oliver on Recurrent Dropsy of left middle Ear—Walton on neglect of Ear-Symptoms in Diagnosis of Diseases of Nervous System—Putman on Examination of Spinal Cord in Polio-Myelitis of Adult—Schmidt on Pathological Anatomy of Cerebro-Spinal Axis of Chronic Myelitis—Fox on two Cases of compression of Spinal Cord by Sarcomatous growths—Seguin on American Method	

	PAGE
of giving Potassium Iodide, and on efficacy of Iodide of Potassium in non-Syphilitic Organic Diseases of Nervous System. By J. A. Ormerod, M.D.	138
Johns: "How the Blind Dream." By Samuel Wilks, M.D., F.R.S.	272
Abstracts of Papers on Diseases of the Nervous System that have appeared in the Guy's Hospital Reports, 1874-1884. By W. Hale White, M.D.	273
De Beurmann on a Diagnostic Test for Sciatica—Hayem and Gilbert on the Changes in the Nervous System after Amputation—Hervouet on the Nervous System of an Idiot. Anomalies in the Convolutions. Arrested Development of the Pyramidal Tract in the Cord—Joffroy on Radial Paralysis: Theory of Nerve-compression—Raymond and Artaud on a Case of Transverse Myelitis—Ballet on Hemiatrophy of the Tongue in Tabes Dorsalis—Raymond and Artaud on Hemiatrophy of the Tongue in Tabes Dorsalis. By J. A. Ormerod, M.D.	282
Pitres and Vaillard on Peripheral Neuritis. By Herbert W. Page	427
Pitres on Anatomico-Clinical Researches on Bilateral Sclerosis consecutive to Unilateral Lesions of the Brain—Fleury on the relations between Ankle-clonus and Knee-jerk. By J. A. Ormerod, M.D.	430
Fol on Micrococcus of Hydrophobia—Horsley and Schäfer on Muscular Contractions Evoked by Excitation of Motor Tract—Hale White on Pathological Histology of Semilunar and Superior Cervical Sympathetic Ganglia. By A. de Watteville	547
Fournier on the Mental Disorders of the Preataxic Stage of Syphilitic Locomotor Ataxy—Legroux and Le Brun on Disorders of Sensation in Cerebral Hemiplegia—Baillarger on the Slaty Discoloration of the Brain in General Paralysis, &c. By R. Atkinson, F.R.C.S.	551
Westphal on the Patellar Reflex in Diagnosis—Remak on the Tendon-Reflexes and the Reaction of Degeneration—Sakaky on Degeneration of Peripheral Nerves in Tabes Dorsalis—Kirchhoff on Localisation of Anovesical Centre—Zacher on Spastic Symptoms in Paralysis—Thomsen and Oppenheim on Sensory Anaesthesia—Bernhardt on Nerve-Stretching in Facial Spasm. By W. J. Dodds, M.D., D.Sc.	557
Demange on Spinal Sclerosis of Vascular Origin—Dumolard on Acute painful Paraplegia—Roger on Tabetic Entorrhœa—Landouzy and Sireday on Left Inferior Facial Paralysis, &c.—Mil's on a Case of Arsenical Paralysis—McNutt on Double Infantile Spastic Hemiplegia—Osler on Jacksonian Epilepsy, &c.—Kemper on a Case of Lodgement of a Brach-Pin in the Brain—Eliot on Poliomyelitis Anterior in Adults—Reichert on the Regeneration of the Vagus and Hypoglossal Nerves—Starr on Cortical Lesion of the Brain—Webber on Multiple Neuritis—Van Duyn on Acute Poliomyelitis in the Adult, and Ranney on a unique Case of Poliomyelitis Anterior Acuta—Seguin on Illustrations of the Anomalous Course of Posterior Spinal Sclerosis—Jacoby on the Neurotic Disturbances after Joint Affections—McGuire on a Case of Nodular Tumour of the Corpus Callosum—McBride on a Case of Tumour in the Optic Thalamus. By J. A. Ormerod, M.D.	561

LIST OF CONTRIBUTORS.

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|---|---|
| ANDERSON, JAMES, M.D. | HAMILTON, PROF. D. J., M.D. |
| ATKINSON, R., F.R.C.S. | HARRIS, THOMAS, M.D. |
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M.R.C.P. | WHITE, W. HALE, M.D. |

LIST OF ILLUSTRATIONS.

	PAGE
Spinal Lesion in Polio-myelitis Anterior Acuta	15
Brachial Monoplegia, Figs. 1-4	80-83
Perforating Tuberculosis of Skull	89
Functions of the Marginal Convolution (Diagram)	100
Anatomy of the Nervous System, Figs. 1 and 2	126, 127
Substitution :—	
Right Hemisphere of the Cerebrum (Diagram)	132
Homonymous Hemianopia	137
Nature of the Objective Cause of Sensation, Figs. 1, 2, 3	191, 194, 207
Cerebral Abscess :—	
Fig. 1. Lateral view of Hemisphere	253
,, 2. Frontal Section	254
,, 3. Pediculo-frontal Section	254
Muscular Phenomenon in Hysteria, Figs. 1, 2, 3	293, 294
Inertia of the Eye and Brain :—	
Fig. 1. Apparatus	298
Figs. 2, 3	303, 307
Ophthalmoplegia, &c. :—	
Temperature Charts 1, 2, 3, 4.	334
,, ,, 5, 6	336
,, ,, 7, 8	337
New Induction Apparatus, Figs. 1, 2, 3, 4	381, 384, 385, 388
Case of Multiple Spinal and Cerebral Tumours (Sarcomata), &c.,	
Figs. 1 to 5	473
Case of Bilateral Degeneration of Spinal Cord, after Hemorrhage in the	
Cerebral Hemisphere	511

B R A I N .

APRIL, 1885.

Original Articles.

CASES OF RECOVERY FROM SYMPTOMS POINTING TO THE PRESENCE OF PROGRESSIVE ORGANIC CEREBRAL DISEASE.

BY J. S. BRISTOWE, M.D., F.R.S.

INTERESTING as all cases of intracranial disease are from many points of view, those are specially interesting to the practical physician in which recovery ensues from symptoms which former experience had led him to regard as indicative of progressive mischief tending to a fatal result. Of recovery, more or less complete, from all kinds of so-called "functional" disorders we have, of course, ample experience. Of recovery also more or less complete, from small hæmorrhagic effusions or from small patches of softening, again clear evidence is sufficiently abundant. Simple inflammation of the surface, or even of the substance, of the brain, no doubt subsides, leaving little or no clinical trace of its pre-existence; and, probably, this is of more common occurrence than most of us suspect. Syphilitic affections, again, are not unfrequently benefited, and sometimes cured, by appropriate treatment. Nevertheless it cannot be denied that symptoms indicative of obvious inflammation of the cerebral meninges, symptoms pointing to the presence of cerebral tubercle, symptoms characteristic of tumours of the brain, and even symptoms apparently referrible to progressive degenerative processes,

are properly regarded as of the gravest omen, and in the great majority of cases foretell a fatal issue.

The cases which I am about to narrate derive their chief interest from such considerations as have inspired the foregoing paragraph. Two of them impressed me greatly; because from their symptoms and progress I had gradually been confirmed in the belief that they were hopeless, when lo! amendment took place, and before long the patients were restored to health.

The first case was that of a young woman, who, when she came under my care, had been complaining for three weeks of abdominal pain; who on her admission into hospital was suffering from subacute peritonitis, with fluid effusion into the abdominal cavity; and who during the next seven weeks presented symptoms which were ascribed, and I think rightly ascribed, to tubercular peritonitis. She had general pain and tenderness of the belly, with more or less fulness, constant sickness, hectic temperature, progressive emaciation and debility, which appeared to be uninfluenced beneficially by any kind of treatment that was adopted. Then she began to squint, from partial paralysis of both external recti, and of the left internal rectus, to present oscillation of the eyeballs, to lose memory, and to have delusions. It was naturally, and I still think correctly, surmised that intracranial tuberculosis had been added to the abdominal tuberculosis; it was also, I think, naturally surmised that the condition of the patient, previously sufficiently grave, had now become hopeless. In the course of a few days, however, to my surprise, the aspect of affairs began to brighten. By degrees, yet rapidly, the temperature became normal, the abdominal symptoms subsided, the sickness ceased, appetite returned, and the patient began to sit up and take interest in all that was going on around her. A little later her squint also disappeared, and she lost her delusions; and six or seven weeks after the first appearance of squint, she left St. Thomas's for a convalescent hospital, still forgetful and still presenting slight nystagmus, but in all other respects perfectly restored to health. I saw her on February 3rd of the present year; she came to see me at my request. I found that for the last twelve

months she had been working at a laundry, that she had had good health, and no recurrence of her former symptoms, and that she looked well, but that she had never recovered her previous good memory. The following is a detailed account of this case:—

CASE I.—*Tubercular peritonitis (?)—Tubercular meningitis, or tubercular tumour of the brain (?)—Recovery.*

ARABELLA D., an unmarried woman 23 years of age, was admitted under my care on the 4th of October, 1883. Her health had been good up to three weeks ago, when, as she was carrying a heavy tray, she felt a sudden sharp pain in the umbilical region. She has suffered from abdominal pain of varying severity ever since; and her abdomen has enlarged.

She is a dark-complexioned, good-looking, fairly nourished girl, complaining of abdominal swelling, and of pain referred chiefly to the hypochondriac regions. The belly is uniformly distended, and measures 35 inches in girth at the umbilicus. It is resonant in front, and dull in the flanks, as she lies on her back; and the relations of the resonant and dull areas vary with position. There is some tenderness on pressure; but no evidence of tumour, or of enlargement of liver or spleen. No anasarca in legs or elsewhere. The thoracic organs are normal, excepting that the heart's apex beats a little higher than natural, and that there is some crepitation at the bases of the lungs. There is no evidence of mischief at the apices. Appetite bad; tongue furred, but moist; pulse 80; respirations 20; temperature varying from 98·4 to 99. Urine, sp. g. 1024; no albumen.

During the next week there was little change in her condition, excepting that the ascites gradually increased, until the girth of the abdomen measured more than 40 inches, and resonance disappeared excepting on deep pressure. The impairment of appetite continued; she complained constantly of pain and tenderness in the abdomen, which were not always referred, as at first, to the hypochondriac regions; her temperature is daily above 100, but varied between 98·4 and 100·6; and her urine was of high specific gravity, and contained abundant urates.

On the 11th she had an attack of diarrhoea, and her bowels were moved eight times; she vomited a good deal, complained of much epigastric pain, and her temperature rose in the morning to 102·2. The tongue was covered with brown fur.

The diarrhoea continued off and on for a week; during the first

two or three days of which she still vomited. She complained during the week of pain and tenderness of the abdomen, chiefly in the epigastric region, and also of pain between the shoulders; her appetite was very bad, her tongue brown and inclined to be dry, and her urine presented a little albumen and a few granular and hyaline casts. The temperature reached 102·8 on the 12th, and subsequently varied between 98·4 and 101. The ascites remained unaltered.

From the end of this week to about the 22nd of November, the patient got steadily weaker and thinner, and suffered from profuse nocturnal perspirations. Her bowels were variable, but on the whole inclined to constipation. She suffered from sickness, and often vomited several times in the day. Her tongue was coated; her appetite very bad. The pain and tenderness in the abdomen never left her; and at times the pain, which was paroxysmal and referred mainly to the umbilical and epigastric regions, was very severe. But during the last fortnight of the time the ascitic fluid disappeared, and the girth of the abdomen became reduced to 27½ inches. The reduction, however, was not attended with any diminution of pain or tenderness, and the abdominal walls became rigid. The temperature varied for the most part between 99 and 101, occasionally becoming sub-normal, and at times rising above 101. From the 13th of November onwards, however, it never reached 100, and was usually normal or sub-normal. The pulse, which was always feeble, ranged from 80 to 116. The urine continued scanty and high-coloured, and usually contained a trace of albumen. The catamenia, which were due about the time of her admission, did not make their appearance either then or subsequently. She had no cough or sign of disease at the apices; but the sub-crepitation, audible at the bases on admission, was audible from time to time subsequently.

On the 24th of November it was noted that for a day or two she had been complaining of double vision; and it was found that she had an internal squint with both eyes (the left external rectus being the weaker), and nystagmus. But she had no headache, giddiness, tremors, or colour-blindness.

On the 26th she was examined by the ophthalmic surgeon, who observed that there was defective movement of both eyes outwards, and slight impairment of inward movement in the left eye, and that the internal structures of the eyes were healthy. There was still nystagmus. Still also she complained of dull aching pain across the upper part of the abdomen; she vomited a little at night; and the urine contained a trace of albumen. Her temperature was normal.

28th.—The condition of the eyes is unaltered ; and there is still some pain across the epigastrium. But the patient has been improving in all other respects during the last few days ; she is bright and cheerful, and hungry ; sickness has ceased ; and her abdominal uneasiness has so much diminished that she has been able to sleep without the hypodermic injections of morphia, to which she has been accustomed almost ever since admission. Pulse 114 ; temperature normal ; urine 1030, many oxalate of lime crystals, and a trace of albumen. Ever since the eyes have become affected the patient has suffered from loss of memory (cannot recollect the days of the week, the times of my visits, whether she has had her dinner, &c.) and from delusions (such as that her mother has brought her a pineapple ; that her father, who has long been dead, has been sitting beside her).

From this time forwards there was progressive improvement. The patient had no return of sickness, and enjoyed good appetite ; her abdomen became more and more flaccid, and free from pain, which finally disappeared wholly ; the diplopia gradually diminished, and had quite subsided by the 16th of December, but nystagmus (especially when she looked to the extreme left) continued ; her temperature ranged from about 97 to 98·2 ; she put on flesh, recovered strength, and became lively and happy. On the 2nd of December, and for a few days subsequently, she complained of pain across the forehead, and slight giddiness ; but these symptoms did not recur. The fundi of the eyes, before she left the hospital, were carefully examined by Mr. Nettleship, who failed to discover any choroidal tubercles, and reported them as being quite healthy.

On the 9th of January she was sent to a convalescent hospital. At that date she expressed herself as feeling, and looked, quite well ; she was enjoying her food, and helping in the ward ; the abdomen was soft and void of pain, tenderness, or tumour ; the urine was free from albumen ; she had no headache or double vision ; but slight nystagmus was still observable when she looked out of the corners of her eyes ; and she was still forgetful. There was no evidence of pulmonary disease. She weighed 6 stone 11 lb.

During her stay in the hospital she was treated mainly with morphia, administered subcutaneously, and tonics.

There is, no doubt, much that is difficult to understand in the narrative which has just been given. It is especially difficult to comprehend how or why the abdominal symptoms should have subsided just as the cerebral symptoms came on, and how or why the latter should in their turn have passed

away, leaving the patient apparently healthy. I think that no one who watched the case during life doubted, and that few of those who read the notes carefully can doubt, that the girl was suffering from tubercular peritonitis. The symptoms and progress of the case were exactly what one constantly witnesses in that disease, and, so far as I know, in no other. Moreover recovery from the symptoms of tubercular-peritonitis, though certainly rare, is not unprecedented. But if she had tubercular peritonitis, it is difficult to believe that her cerebral symptoms could have been due to anything else than intracranial tuberculosis. I do not see how the combination of double internal squint, nystagmus and loss of memory, could have been merely functional. There was no ground whatever, from the antecedents of the girl, to suspect syphilis; moreover she recovered without the use of antisiphilitic treatment. No doubt inflammation at the base of the brain may be idiopathic; and this might furnish the explanation of her symptoms. But the arguments which might be adduced against the presence of simple meningitis are of the same kind as those that might be adduced against the presence of tubercular meningitis; and it is the less likely of the two explanations in this case, inasmuch as there was strong evidence in favour of the presence of tubercles elsewhere. I believe that the patient was suffering from cerebral tuberculosis; but whether this was in the form of slight basal meningitis, or of tumours in the substance of the cerebellum or elsewhere, I cannot venture to decide. The absence of optic neuritis, which is not uncommon in either of these affections, does not help the diagnosis.

In connection with this case I may briefly advert to another which came under my notice some years ago, and which has some kind of relation to it.

CASE II.

On the 5th of May, 1875, a servant-girl, 15 years of age, came under my care. She had had good health up to three weeks before I saw her. Then she began to suffer from pains in the head, back and abdomen, giddiness, drowsiness by day and restlessness at night. Also she began to see double.

On admission, she was a fairly healthy-looking girl, complaining

of headache, giddiness and double vision. She was very drowsy, and indisposed to answer or take notice. Her pulse was 48, her respirations 24, her temperature normal. She had paralysis of both external recti, and well-marked double optic neuritis of recent origin. For some days afterwards she remained somewhat drowsy and torpid, with slow pulse, and at times irregular respiration of the Cheyne-Stokes character; her temperature at the same time being slightly below the normal. Then she gradually improved, her squint disappeared, and at the end of four weeks from her admission left the hospital well, excepting for the persistence of optic neuritis.

She was re-admitted twelve days later, suffering from weakness, pains in back, short breath on exertion, and slight cough. She also complained of a little pain in the right temple, weakness of eyes, and some giddiness when walking. She was suffering from slight bronchial catarrh, and while in the hospital had an attack of erythema nodosum, during which her temperature varied between 99 and 102, and on one occasion rose to 102·8. She remained in the hospital two months, and left fairly well. On leaving, there was still marked evidence of double optic neuritis but no other definite indications of cerebral disease. There was no clear indication of tuberculosis. But I thought at the time, and am still inclined to think, that her symptoms were due to tubercular meningitis.

The third case was that of a woman, 39 years of age, who in the midst of apparently good health was attacked with double vision and giddiness, followed in about three weeks by headache and inability to stand. Then she vomited, and complained of numbness of the right half of the upper lip. At the end of a month she had headache, staggered like a tipsy person when she had attempted to walk, suffered from distressing nausea, had incomplete paralysis of the right portio dura, paralysis of the right external rectus, and slight paralysis of the left internal rectus, horizontal nystagmus when she turned her eyes strongly to the left, and contraction of the right half of the field of vision. She had no other paralysis, no impairment of sensation, no affection of the internal muscles of the eyes, no optic neuritis, no colour-blindness, and no loss of taste or smell. Soon afterwards the left external rectus became paralysed. Then came numbness and tingling in the feet, with involuntary jumpings of the legs, tendency to

stiffness, and increase of tendon reflexes. A short time afterwards a little twitching of the left angle of the mouth was observed, which soon extended to the left eyelids and to the left hand. By this time too she had become colour-blind with both eyes. Lastly, numbness and weakness of the right arm supervened, followed by marked contracture. At the end of three months from the beginning of her illness, her symptoms were at their worst; and, looking to their gravity and their rapid progress, I must confess that I took a very unfavourable view of the patient's chances of recovery. Nevertheless amendment took place; one by one her symptoms disappeared; and in two months she left the hospital in all essential points restored to health. And now at the end of nearly two years she is, I believe, earning her livelihood as a nurse. The following is a full account of this case.

CASE III.—*Symptoms pointing to progressive disease in the neighbourhood of the fourth ventricle, coming on gradually, and finally subsiding under treatment.*

ELIZA N., a nurse, single, aged 39, was admitted into St. Thomas's under my care on the 17th of January, 1883.

Excepting that she had had an attack of enteric fever ten years previously, and subsequently occasional slight rheumatic pains, she had enjoyed excellent health, until her present illness began. There was no history or evidence of syphilis.

She had been attending on a private paralytic patient, when suddenly a month ago, while feeding him, she was attacked with double vision. This was accompanied by a sense of giddiness and nausea—a feeling of sea-sickness, as she termed it. These symptoms continued, and about three weeks afterwards she found on getting out of bed that she could not stand, and in fact tumbled while stooping for her slippers. On the 12th she first complained of headache over both the frontal and the occipital regions, a sense of pressure, and as if her head were too heavy for her. On the 15th she vomited, and noticed numbness and weakness of right half of upper lip.

She is a healthy-looking, well-nourished woman, complaining of headache, nausea, giddiness, and consequent inability to stand, and difficulty in using the upper lip on the right side. Her headache is severe and more or less general, but is referred mainly to the frontal region. There is an area of tenderness to percussion,

however, at the back of the left parietal bone. The forehead also is somewhat tender to percussion. The nausea is distressing, but is present mainly when she sits up in bed, or tries to stand. She is unable to walk without assistance, or even to stand; staggers like a drunken person, and has a tendency (she says) to fall over to the left rather than to the right side. There are no ataxic movements. There is slight, but obvious, paralysis of the right facial nerve; the right eyelids close imperfectly; the right upper lip evidently acts feebly; and the right side of the face generally is smoother than the other. Nevertheless, the right angle of the mouth moves freely when she laughs. The tongue is protruded slightly towards the left, but when the organ is drawn in again its left side looks plumper and lies higher than the right. There is no obvious difference in the appearance or action of the two sides of the soft palate; but the uvula is concave towards the right side, and its apex points in that direction. She presents a marked squint. The right external rectus appears to be completely paralysed; and the left internal rectus slightly paralysed; and there is well-marked horizontal nystagmus when she looks strongly to the left. Pupils normal. There is no optic neuritis. She can distinguish the forms of objects and colours perfectly; but there is apparently some contraction of the field of vision towards the right side. Smell and taste and speech are unimpaired. No affection of ears, excepting that she has slight deafness on the left side, which dates from childhood. No paralysis or anæsthesia of limbs; tendon and superficial reflexes normal; mental condition healthy; no hysterical symptoms. Thoracic and abdominal organs healthy; tongue clean; appetite fair; bowels open; catamenia regular; urine normal; temperature 99·6.

The presence of the symptoms above detailed were confirmed during the next few days by repeated and careful examination. And Mr. Nettleship not only concurred in the description of the eyes above given, but, by investigation of the field of vision, discovered that the right half was so largely contracted for both eyes, as almost to cause hemiopia. Her headache varied in severity, but was rarely if ever absent, and often intense. Her nausea remained for the most part in abeyance so long as she lay perfectly still, but became severe whenever she sat up in bed, and especially when she was made to stand. Her appetite was maintained. She continued quite unable to stand alone.

On the 23rd it was noticed that there was decided weakness of the left external rectus, in addition to the former ocular defects. This became more pronounced during the next four days.

On the 27th she first experienced a feeling of numbness and

coldness at the bottom of the feet, which in the course of a few days amounted to a sense of pins and needles. On the 12th of February she complained that her legs felt stiff, and that they jumped at times. She could move her legs pretty freely; but they tended to become rigid, especially at her knee- and ankle-joints; there was marked exaggeration of the tendon reflexes, and on the left side ankle-clonus. These phenomena continued for a time, the left leg being worse than the other. Ankle-clonus was obtained later on the right side, which also was somewhat more numb than the other.

On the 3rd of February it was observed that the facial paralysis had increased; although when laughing the right angle of the mouth was still drawn up, at least as much as the left; but it was also noticed that there were frequent twitchings of the left angle of the mouth. These twitchings continued; and about the 20th it was noted that she had also occasional twitchings in the left eyelids, and slight tremors in the left hand.

She had complained for a short time that her eyesight was not so clear as it had been; and on the 1st of March this was again carefully tested. At that time the ocular paralyses remained; the fields of vision were as before; and the discs were quite clear, but doubtfully pale. But now she was colour-blind. She could not distinguish greens or reds, and confounded them with brown, and sometimes with grey. Bright yellow was called white. Bright blue and lilac were both called dark blue. She was sure also that her vision was in other respects worse than it had been.

On the 30th of March, after she had been suffering for a day or two from much more intense pain than usual, she for the first time complained of a feeling of pins and needles in the right hand and arm, and of pain in the right shoulder. The arm also became weak, and in the course of a week or two slightly flexed at the several joints, the fingers especially suffering; and she had more or less pain from the shoulder downwards. This paralytic affection of the arm was never complete; but attained its maximum towards the end of April.

During the greater part of the time terminating with the last date, the patient had on the whole been getting slowly but steadily worse; the pain in the head (which varied in position, but became more and more localised in the neighbourhood of the back part of the left parietal bone, as time went on) was constant, but liable to severe exacerbations; the sense of nausea on the slightest movement continued; and from time to time (as has been shown) additional paralytic phenomena arose. Nevertheless during the month of April some favourable indications were manifested. On

the 11th she saw singly for the first time since admission. And on the 15th the following note was taken: "Has not seen double since the 11th, and now the movements of the eyes appear to be perfect. She distinguishes colours better than she did a little while back; she recognises bright greens and blues, but calls red black, and yellow dirty white. There is still nystagmus when looking to the extreme left. Also there seems some improvement in the facial palsy; at any rate she closes her eye better than she did, and says that the right side of the face is less stiff. Complains that left side of head is heavier than right." From this time forward there was no return of double vision; and her power of appreciating colours was slowly restored.

Late in April, or early in May, the patient began to improve decidedly. She still suffered from intense headache, giddiness, and nausea; but the attacks were not so frequent; and intermissions occurred which became longer and longer. Her appetite was better, and she felt stronger. About the 10th or 11th of May she began to sit up for a few hours in the evening; and a week or two later got up in the afternoons, and even began to walk about with assistance. It remains only to add, that her various paralytic symptoms gradually cleared up; and that, when she left the hospital on the 4th of June, there were scarcely any traces of nausea or headache, the right facial nerve was not visibly paralysed (although she said that side of the face was still stiff, and that there was still a little difficulty in retaining fluids within the lips), the right hand and arm had almost completely recovered their power, and could be used freely, there had been no starting in the legs for some weeks, and (though the right leg was still somewhat stiff) she could walk well.

She went for a month to a convalescent hospital; at the end of which time she presented herself for examination. She then seemed quite well, and expressed herself as being able to resume her occupation. There were no discoverable signs of paralysis, and her appreciation of colours was entirely restored. I saw her some months later, and she remained well. I may add that there was still a little twitching about the left angle of the mouth. I think it probable, however, that this was an old affair.

During the greater part of the patient's illness her temperature varied between 99° and 100° ; but occasionally it rose to between 101° and 102° , and more frequently descended to the normal. I cannot say that it improved materially as her condition improved in other respects.

The treatment adopted was mainly the subcutaneous injection of morphia, repeated often two or three times a day, for the relief

of headache; and the occasional use of leeches behind the ears (the application of which was usually followed by benefit), of blisters and of ice.

For the first week or two I prescribed 5 grains of iodide of potassium, and 40 minims of solution of perchloride of mercury, to be taken three times a day. Then this was replaced for a time by bromide of potassium, in 20-grain doses. Then she was treated, for reasons not referred to in the above notes, at one time with some stomachic mixture, at another time with some cough mixture. The iodide of potassium and mercury were resumed on the 15th of April, and continued until she left.

What was the matter with this patient? Had she a tumour of the brain? Many of her symptoms—her localised headache, giddiness, nausea and rapid extension of symptoms—suggested this explanation. But the absence of optic neuritis and her final recovery seemed to negative this view. Was her disease a functional one merely? I think a decided “no” may be answered to this question. There was no history of hysteria; and she was not at all emotional. Moreover the character of the symptoms and their mode of development were not in accordance with one’s experience of mere functional disorder. That her symptoms could not have been due to obstruction of arteries with consequent softening of some limited tract of brain-tissue, or to hæmorrhage, is clear from the fact that their development extended over three or four months. In many respects her case presented a close analogy to cases of ophthalmoplegia, presumably due to degenerative changes, or chronic inflammatory processes. In these we not unfrequently observe headache, giddiness, sickness, and (besides the paralysis of the eye-muscles) various other paralyses, anæsthesia of limited distribution, and involvement of one or more of the special senses, without optic neuritis. But these cases, so far as I know them, are of much slower progress than hers, and do not tend to recover. I do not see, however, why such cases should not occasionally improve or even recover; and on the whole I lean to the opinion that, in this patient’s case, the symptoms were really due to some subacute progressive inflammatory process taking its origin somewhere about the floor of the fourth ventricle, and spreading thence in depth and surface.

Her recovery under the use of iodide of potassium and mercury suggests a syphilitic origin to her symptoms. I never dared myself to ask her whether she had had this disease. She was a very healthy-looking woman ; there was no lump or scar or stain about her body to suggest that she had ever had anything of the kind ; and her demeanour was such as to disarm suspicion. Nevertheless the possibility of the affection being syphilitic cannot be altogether excluded from consideration.

ON THE NATURE OF THE SPINAL LESION IN POLIO-MYELITIS ANTERIOR ACUTA, OR INFAN- TILE PARALYSIS.

BY DAVID DRUMMOND, M.A., M.D.,

*Physician to the Infirmary, and Children's Hospital, Newcastle-on-Tyne ;
Joint Lecturer on Pathology, University of Durham College of Medicine.*

IT is the opinion of well-nigh all pathologists that the paralysis and atrophy so typical of this disease are the result of destruction of the ganglionic cells in the anterior grey cornua. This is the position taken up by nearly all clinical and pathological teachers, without any fear of contradiction; and, indeed, numerous post-mortem examinations, made in cases where death has resulted from some independent affection, some time after the onset of the lesion, have proved its truth. It has, however, fallen to the lot of very few to examine the cord of a child very soon after it has become affected, in what might be called the acute or inflammatory stage, for, as is well known, it is a lesion that very rarely proves fatal; consequently the exact nature of the essential and primary lesion is little more than a matter of conjecture. Such a chance has recently occurred to me—for which opportunity I am indebted to my friend Dr. Pope, of South Shields. The child whose spinal lesion I am about to describe was a girl aged five. At breakfast-time of the day of her death she was in her usual health, but shortly after her meal she felt sick and vomited. She was then put to bed, when she slept for several hours. Early in the afternoon it was observed that she was feverish and evidently in a dying condition, an observation that was soon consummated, as her death took place six or seven hours after the commencement of her illness—the result, as was afterwards surmised, of respiratory paralysis. At Dr. Pope's request I made a post-mortem

examination. The body was well nourished. The heart was healthy. The lungs were somewhat congested, and in the left was found a small nodule of catarrhal pneumonia, about the size of a walnut. The abdominal cavity and its organs were normal. The brain appeared to be healthy in every respect. Failing to account for death by the changes noted in the lungs, which, so far, were the only morbid conditions detected, I proceeded to examine the upper part of the spinal cord, when the usual transverse sections at various levels from the medulla oblongata downwards brought to light an altered condition of the anterior grey horns between the third and fourth cervical



nerves, the appearances at the level of the latter suggesting *red softening*. This portion of the cord was hardened in a solution of bichromate of ammonium, cut with an ice-freezing microtome, and stained with carmine.

When examined by the low power of the microscope (half-inch), the extreme vascularity of the anterior horns was at once apparent. In some of the sections, in addition to the unusual display of capillaries, minute patches of yellow figured prominently upon the carmine groundwork. The ganglionic cells were seen to be numerous and distinct, though apparently swollen. With the high power ($\frac{1}{6}$ inch, see figure above), the

blood-vessels, distended with corpuscles, formed a striking feature, not only of the anterior horns, but of the anterior white columns and middle and anterior portions of the posterior horns as well, for the inflammatory mischief was by no means confined to the first-named region. Following the tracks of the arteries (internal and external anterior root arteries, and the antero-lateral branch) as they made their way from the front of the periphery of the cord, through the white matters to enter the anterior horns, minute hæmorrhages were scattered here and there, whilst congestion, with free leucocytes, was apparent. In places the neuroglia was swollen and so altered as to obliterate in part the meshy character of the structure, and thus conceal the cut ends of the axis cylinders. In this way the bulk of the anterior columns formed a marked contrast to the beautiful and distinct arrangement of neuroglial network, with the nerve fibres seen as central dots, which marked the postero-lateral columns. As the anterior grey horn was approached, the signs of inflammation increased, attaining a maximum as the swollen and somewhat ill-defined mass of grey matter was reached. Here the dilated vessels were crammed full of corpuscles, whilst the capillary network was rendered most distinct, each vessel containing its single or double row of cells. The minute hæmorrhages, which were seen as collections of yellow-coloured blood-cells, formed the most striking feature of each field that was examined. These extended as far back as the distribution of the posterior branches of the central artery on each side, that is, quite into the posterior horns, but they were most numerous in the anterior cornu, about the ganglionic cells. The large cells formed prominent objects, though they were apparently swollen, granular, and rather ill-defined; and it was observed that the majority had lost their nuclei, though here and there a ganglionic cell with a nucleus could be distinguished. Their processes were no longer visible, and they appeared to fill completely the spaces in which they lay. The cells that presented the most obvious changes were more or less surrounded with dilated and blocked capillaries and minute hæmorrhages; some, indeed, appeared to be imbedded in blood-corpuscles.

In most of the sections it was apparent that one horn had suffered more than its fellow, though in most of those examined both horns showed inflammatory changes. The grey fibres which form the bulk of the anterior horns were swollen and obscure, the whole presenting an indefinite and somewhat granular appearance, which contrasted strangely with the uninjured portions of the posterior horns and roots.

The pronounced changes above described were only observed in a very limited portion of the spinal cord, for as the sections gradually ascended or descended from the affected area, a return to the normal state was brought to light, the signs of inflammation diminishing in intensity, until a few isolated hæmorrhages with distended capillaries alone were found.

Although the accident of the situation of the lesion, attacking as it did the region of the origin of the phrenic nerve, prevented the little patient from living long enough after the onset of the inflammation to admit of an exact clinical diagnosis, I do not imagine that any one will be found to question the identity of her case with *polio-myelitis anterior acuta*, and I make no apology for the assumption that they are the same. I think, therefore, that the case is one of considerable importance, as demonstrating the early changes that lead up to the later degenerations which all are so familiar with in old-standing cases of infantile paralysis.

In the year 1870, Dr. Clifford Allbutt recorded in the 'Lancet' ¹ a case which has been frequently quoted by writers on infantile paralysis (Ross and others), and I think erroneously, as an example of the disease. In this case an examination of the cord, soon after the occurrence of the lesion, revealed two hæmorrhages in the cervical region, "one a small one in the left posterior horn, and the other larger, in the right posterior horn, breaking into the lateral column." I do not think this case can be fairly regarded as a true example of *infantile spinal palsy*, as Dr. Allbutt points out that the paralysis, which affected all four extremities, came on a few minutes after the child, whose age was six months, had been lifted sharply, when its head fell heavily forward. Obviously there is here an element of traumatism foreign to most cases of acute anterior polio-

¹ 'Lancet,' 1870, vol. ii. p. 84.

myelitis of infants; and, further, the hæmorrhages appeared to be confined to the posterior half of the cord, a region which cannot be looked upon as the real seat of the disease, though affected in the case I have described, and also in one to which I am about to refer, recorded by Dr. Angel Money. I am therefore disposed, with due deference to Dr. Allbutt and those who, in quoting the record, have shared his opinion, to think that the hæmorrhages in the case were the result of injury rather than inflammation.

In the 'Transactions of the Pathological Society of London' for 1884 (just published), page 45, Dr. Angel Money describes under the title, "The Spinal Cord of a recent and old case of Infantile Palsy," the appearances of the cord of a child, aged two years, who died of pulmonary disease sixteen weeks after the onset of an attack of acute anterior polio-myelitis. The changes found in the lumbar enlargement were "(1) great distension and thrombosis of vessels, especially in the anterior cornua; (2) infiltration of the cornua with abundant leucocytes; (3) absence of large multipolar or other nerve cells." Dr. Angel Money goes on to state as follows:—"Further, it was seen that the disease was not confined to the anterior horns, but spread forwards, outwards, and backwards; though the principal focus of the mischief was certainly the centre of the anterior horn." This observation, published some time after I had written the above short account of the spinal cord in Dr. Pope's case, confirms my own, in so far as it shows that the inflammation is not by any means confined to the large ganglionic cells, or even to the anterior horns; but the fact that it was made sixteen weeks subsequent to the attack, is sufficient to explain the apparent slight differences between Dr. Money's and my own description of the primary lesion.

Althaus states in his essay,¹ and I believe him to be correct, that there were at that date no *post-mortem* records where the child had not been paralysed for at least two months, nor am I aware that any more recent examinations have been made since he wrote. The case I have recorded would appear to set at rest the question discussed by Charcot, Roth, Dujardin, and others, namely, whether the inflammation is

¹ 'On Infantile Paralysis.' London, Longmans, 1878.

parenchymatous or interstitial, for it is obvious that the several parts affected—the neuroglia, the nerve fibres, and the ganglionic cells—must take part, though they may not all share alike, in the destructive processes induced by the inflammation. Indeed, the brunt would appear to fall on the large cells, a fact revealed alike by the examinations of old-standing cases, and the clinical features of the disease.

Reference has already been made to the extension of the inflammation beyond the limits prescribed by the various writers on the subject, for it will be recollected that though the fresh changes were found most abundantly in the anterior horns, they, nevertheless, affected the antero-lateral white columns, and part of the posterior horns as well; in fact, the anterior cornua were only the centres of inflamed areas, which reached considerably beyond their limits. It will, therefore, become a question whether the name proposed by Kussmaul, and adopted by the majority of writers—*anterior polio-myelitis*—is really an appropriate one, though usage in the nomenclature of disease, as in other matters, will be found to resent interference.

In conclusion, I would like to point out two ways in which the recognition of the real nature of the primary lesion of infantile paralysis is of importance. In the first place, it throws light upon the attacks of *temporary spinal paralysis of infants*—seizures, if I may be allowed the expression, which I confess have puzzled me not a little. An example, briefly related, may not be out of place. A little boy, aged 16 months, whilst cutting his eye-teeth, was observed one day to be unusually peevish when being dressed to go into the garden in his perambulator. As he continued to fret, his nurse attempted to amuse him by offering him a ball, when she discovered that he could not raise the left arm to seize it. He was at once taken into the house and undressed, in order to discover if the arm had been injured, when it was found that he was feverish, and that the left arm was very hot and painful (hyperæsthetic). There was no bruising or other evidence of injury, but the limb hung down completely paralysed. When asked to clap his hands, an accomplishment which he had acquired some time before, he contented

himself with slapping his right hand against his leg, ignoring the left entirely. The paralysis lasted for 24 hours, and recovery of motion, which was apparently accelerated by the application of cold to the limb, was first observed in the fingers.

It will not be difficult to explain such an attack by the supposition that a limited area of the left anterior horn in the cervical enlargement had suddenly become congested, the lesion stopping short of actual inflammation.

In the next place, the necessity for early energetic anti-phlogistic treatment is emphasised, in order to overcome the inflammation, or reduce it as much as possible, by rest, cold to the spine and affected limb, leeches, or, as recommended by Althaus, the hypodermic injection of ergotin; and the fact that some attacks are only temporary, whilst in others, and indeed nearly all, there is a tendency to recovery shown by some of the muscles at first paralysed, should stimulate us to endeavour to recognise the condition as early as possible, and to carry out thoroughly the curative means at our disposal.

ON SOME CENTRAL AFFECTIONS OF VISION.

BY W. J. DODDS, M.D., D.SC.,

Montrose Royal Asylum.

THE labours of Ferrier, Munk, Goltz, and others have done much during the last few years to advance our knowledge of the sensory functions of the brain, especially of the cerebrum. They have used the experimental method, and their conclusions apply primarily not to man, but to the monkey, dog, and other lower animals.

The spectacle of doctors disagreeing is one that many people can observe with equanimity, and some few, unfortunately, with unaffected pleasure, especially when the doctors are vivisectionists, and the moral can be so easily pointed. In this question of the functions of the cerebrum, the results are no doubt differently interpreted by Ferrier and Goltz. It would be strange if there were not differences in so complicated a problem as that of the functions of the most highly organised of matter. But putting aside the rival interpretations, there still remains to us the solid store of facts which these observers have accumulated, and the influence of which is already perceptible in the fresh impetus given to the clinical and pathological investigation of the functions of the human brain. It is to this clinico-pathological aspect of the question that we propose to direct attention in this paper.

A few years ago Ferrier, searching the records of cerebral disease for mention of affections of sight, hearing, and the other senses, met with such poor success, that he concluded the latency was not a latency of symptoms but of observation. The justness of this criticism is indisputable, and the recent progress of this department of medicine has amply vindicated it. To take the affections of sight as an example; the last

decade has seen marked advance in our knowledge of amblyopia, hemianopsia, "psychical blindness," and word-blindness; and we find that in certain cerebral diseases affections of sight instead of being rare occurrences, are as frequent as the occurrence of aphasia.

There are many ways in which the problem of the functions of the brain and their localisation may be approached by the physician. We shall limit ourselves here to the consideration of sight and its connections; and we shall find it convenient to divide the subject into two parts; the first treating of sight and its associations, or, to adopt a more physiological phrase, visual reflexes, using the word reflex in a very broad and perhaps strained sense; the second, treating of the localisation of these reflexes in the brain.

Visual Reflexes.

Much confusion has arisen from the introduction of the terms of a subjective psychology into the study of objective brain physiology. Writers have puzzled their readers, and often we fear themselves, in their attempts to define consciousness, and distinguish between sensation, perception, apperception, cognition, and the like. These abstract distinctions have their value and their place, but we must not forget that they are merely stations that a subjective psychology has made, not found, on the unbroken line of a continuous nervous process. It will be our endeavour to banish these terms as far as possible from our inquiry, and to proceed as objectively as we can.

Stated briefly, our aim is to note the effect produced on a person by the sight of different objects. It may be that light, and light alone, has influence on him, and that the contraction of the pupil is the only reflex that can be roused; or he may notice persons and things, and follow them blankly with his eyes; or he may grasp at objects which are offered him, and yet not know what they are, or how to use them. A still higher development may be reached, and the sight of food may suggest the movements requisite for eating; well-known faces may be recognised, writing understood, or the most complex visual pictures and symbols appreciated and acted on.

It is of course absurd to think of enumerating all the visual reflexes that are to be found even in a man of the meanest intellectual endowments. The most laborious lexicographer would shrink from the task of compiling a table of contents of even this one department of the human mind, though there is certainly a limit to its contents. We can, however, divide the visual reflexes into certain great classes, and arrange them in an order advancing from the simple to the complex, from the completely organised to the least organised, from the stable to the unstable, preserving, in fact, as far as possible, the order of their evolution, the inverse of which is the well-known order of dissolution or regression upon which Herbert Spencer and Ribot lay such stress in their writings.

Such a classification will demand a wide and careful investigation. For this purpose the dawn of the visual reflexes must be observed in infants, and such observations as Darwin's and Preyer's pressed into service. In the same way the gradual lapsing of the reflexes in the decay of age will be of use. The phenomena of sleep, somnambulism, mesmerism, automatism, will all yield valuable information, which can be supplemented by an examination of the state of the reflexes in the different stages of the action of anæsthetics, and of toxic agents such as alcohol. Then, too, the field of observation must include the visual reflexes in idiocy and imbecility; in the peculiar mental states that follow epileptic and paralytic attacks; in word-blindness and the allied affections; and in that most complicated group of all, insanity.

One of the chief difficulties in satisfactorily grouping together the visual reflexes, and arranging the classes in an order advancing from the simple to the complex, lies in the fact that the simplest and most direct reflexes are, by the clustering of associations around them, gradually raised into the position of complicated and involved reflexes. The retino-muscular reflexes, for instance, that are concerned in focal and axial adjustments, and that are associated with the idea of distance, are among the simplest of the reflexes, and are developed in our earliest childhood. For example, a child soon learns only to grasp at what is approximately within reach. But these same reflexes in the mariner, the sportsman, the land-

surveyor, are by a process of education associated with much juster ideas of distance, and their suggestions are checked by a variety of other recollections. Thus, although these retino-muscular reflexes are the basis of all ideas of distance, yet it would not be a natural classification if we regarded all judgments of distance as contained within this elementary group. To take another example: there are certain protective visual reflexes that form a very convenient clinical group. In this group we include such acts as blinking, when a finger is thrust up to the eye; starting, if a sudden stabbing movement is made with a knife; aversion, if a red-hot poker is offered, or if a cup of steaming water is held to be drunk out of. These reflexes are based on some of our earliest and most powerful impressions, and they constitute a group of what may be called low and well-organised reflexes. But it is difficult to formulate an accurate definition of the group, for it gradually shades off into higher reflexes, based on less frequent or less powerful impressions, and exhibiting greater intellectual action. This is a difficulty that is felt in all the groups, but it must not discourage; it is felt in all classifications, for nature abhors sharp distinctions and dividing lines as much as she does a vacuum. Another difficulty is the order in which these groups of reflexes should be arranged. The evolution of individuals differs within great limits, and reflexes that are firmly organised in one are incompletely organised in another. The groups that we offer are merely provisional, and are put forward as an indication of the line on which we ought to proceed in building up the reflexes, and more especially as a guide in the examination of cases; for it is by such a regular and orderly examination of the reflexes in connection with sight, hearing, and all the senses, and by endeavouring to distinguish the different associations that are concerned in them, that we may hope to arrive at a satisfactory knowledge of the mental condition of a patient.

There is need of a word of caution here against the error of concluding that a reflex is lost because it is not immediately forthcoming. It may be held in check and prevented by some inhibitory power. What we call the will may from sheer contrariety set itself to counter-work the ordinary reflex

actions, or it may be urged to this course by powerful motives. Again, the attention may be so concentrated on some particular thought, that the visual impressions die away, and lack strength to call forth the usual reflex; the fixed ideas of certain forms of insanity furnish us with the best illustration of this variety, which also includes cases of abstraction, reverie, dominant ideas, &c. Or it may be that the reflexes, especially the higher ones, are not obtained, because of the want of attention. It is important to remember and avoid these sources of fallacy, in deciding on the condition of the reflexes.

We can imagine objectors saying, "Well, suppose you get the so called visual reflexes properly grouped together, and something like a natural classification formed, of what use will it be? You are not dealing with sight alone; in your visual reflexes there is a regular jumble of sight, touch, hearing, the kinæsthetic sense, in fact of all the senses, with a few of the appetites, several distinct instincts, and the whole group of the higher faculties of reason, memory, judgment, attention, &c. —such a jumble, in fact, that it is quite impossible to say where sight begins or where it ends, or in some of the reflexes to see what sight has to do with them at all."

With most of this criticism we should cordially agree. We are not dealing with sight alone, with sight defined as sensitiveness to light, or mere ability to see form and colour. Sight has in these brain questions no such restricted meaning. It is recognised that there is sight and sight, and we speak of intellectual or psychical blindness, word-blindness, and other forms of blindness. We have, in a word, to deal with sight and its associations; with the visual pictures that are called up by the sight of an object as well as with the tactile, auditory, motor (or, as Bastian would say, kinæsthetic), and other memories that are aroused. Such an examination is, unfortunately, fraught with complexity, but complexity is inherent in the problem. The various senses are knit together in the brain in a network of associations, the plan of which we can never hope fully to know; but we shall do better if we work at the fringe of this network, and seek to obtain a knowledge of it rather than weave for ourselves a new fabric, and delude ourselves with the idea that it is nature's product. The method

of the visual reflexes has been selected, not because it furthers any *à priori* notions of well-defined cerebral localisation, but because it provides us with a satisfactory view of one department of a patient's mind. The splitting up of these reflexes into their component parts, and the determining in cases where they are absent, whether the affection is on the motor side, or the sensory, and in the latter case the deciding as to how far the appearances are due to disorder of the purely visual associations, or of the associations with other senses—these are problems of varying difficulty in different cases. Sometimes they are comparatively easy of solution; at other times it is impossible to arrive at a satisfactory conclusion, owing to the condition of the patient, or to our present lack of knowledge. Take, for example, the reflex of reading at sight. Does the sight of a word call up the sense at once (by recalling we will say, the picture of the object), or only after arousing the corresponding auditory sound; or, yet again, only after arousing a suppressed articulation of the word? Until these points are settled, we cannot hope to analyse satisfactorily cases of word-blindness.

Before passing to a consideration of the various groups of reflexes, we would again emphasise the importance in case-taking of being dissatisfied with such general terms as hebetude, stupor, dementia, enfeebled intellect, impaired faculties, &c. We should describe the facts that come before us, as far as possible grouping them around the senses they primarily concern. We would also note the practical importance of investigating the condition of the eyes separately as well as in combination.

We shall in our view of the visual reflexes confine ourselves to what may be called presentative sight as distinguished from re-presentative sight; in other words, we shall occupy ourselves with the visual associations that the sight of an object calls forth, and not with those that are called forth by auditory or tactile impressions, or by the internal rehearsing of a name. We shall further limit our attention chiefly to those conditions implying paralysis or loss of function, and shown by absence of the visual reflex—leaving untouched the consideration of those more complicated conditions characterised by perversion

or exaggeration of function, excluding, therefore, hallucinations, and illusions of sight. The examples we shall give, particularly those in sections 2 to 8, are taken mainly from observations made by Fürstner,¹ Reinhard,² Stenger,³ Zacher,⁴ and ourselves. We do not deem it necessary to give more than these general references.

We premise that in the cases examined there is no purely optical defect, no defect of media, of the retina, or of the optic disk.

(1.) *The Light Reflexes.*—Under this head we may conveniently include three phenomena:—the contraction of the pupil when a bright light falls on the retina; the dilatation of the pupil when the eye is removed from a bright to a dull light; and the emotional effects of light. The stimulus may be pleasurable and cause a smile, as is often seen in infants; or it may be too powerful, and be followed by an expression of pain, movements of general uneasiness, closure of the eyelids, aversion of the head, and raising of the hands, to the eyes.

These three phenomena should be investigated separately. The contraction of the pupil to light is the lowest and best organised of the visual reflexes; its relations to the other reflexes of this group and to the higher reflexes are not so clearly made out as is desirable. The pupil may contract to light, and yet there be no emotional manifestation of pain or pleasure, even though a strong light is shone into the eye; and conversely, the latter may be present and the former absent. Another point to be considered is the possibility of a retinal impression stimulating higher centres that inhibit this lower reflex. In studying these reflexes we must remember to exclude the contraction of the pupil that occurs on convergence of the eyes; the pupil may contract well on convergence, but badly to light.

(2.) *Objects fixed and followed by the Eye.*—The characteristic of this group of reflexes is that the eye does not gaze vacantly

¹ Fürstner, 'Archiv f. Psych.' viii. p. 162, and ix. p. 90.

² Reinhard, 'Archiv f. Psych.' ix. p. 161.

³ Stenger, 'Archiv f. Psych.' xiii. p. 218.

⁴ Zacher, 'Archiv. f. Psych.' xiv. p. 489.

into space if an object is held before it or crosses its field of vision, but the object is looked at and its movements followed; it is seen.

The fixing of an object by the eyes is by no means the simple act that it appears. Leconte points this out with great clearness in his work on Sight, and he shows that in every act of looking at an object, there are involved, besides the innate idea of direction, and the principle, almost as innate, of corresponding points, three adjustments,—the focal adjustment, whereby each eye obtains a perfect image of the object; the axial adjustment, whereby the axes of both eyes meet in the object; and thirdly, the pupil-adjustment, seen in its contraction on convergence of the eyes. Now each of these adjustments is based upon a visual reflex, so that we have the retino-ciliary reflex, the retino-ocular reflex (as we may term the arc that connects the retina with the ocular muscles), and the special retino-iridal reflex. Further, these adjustments or reflexes are grouped together and co-ordinated in the act of looking at an object, and with them are associated certain movements of the head and neck. Each of these several reflexes may be disturbed, and also the group of co-ordinated reflexes; but these are affections that belong to the domain of the ophthalmic surgeon, and we shall do no more than mention them. Looking at things and following them with the eyes gives rise to ideas of form, size, and distance; indeed we may say that these ideas are present potentially or actually in every visual picture. Are there cases in which these fundamental ideas are seriously affected? It is only in a few exceptional cases that we are able to arrive at a conclusion. Infants who have not commenced to check their visual by their tactile experiences have the vaguest ideas of distance, and Cheselden's celebrated patient saw things flat as in a picture, and said that all objects seemed to touch his eyes. We are not aware of any observations on the adult which indicate an affection of these fundamental ideas.

Let us now consider the cases in which this power of seeing objects is impaired or lost. We need scarcely say that the retina is a surface, not a point, and that consequently sight may be lost in one part of the retina, and retained in another.

This leads us to affections of the field of vision, and of these hemianopsia is the most important.

We can only draw attention to a few of the main points in connection with *hemianopsia*. We should in every case determine, by the perimeter if possible, whether vision is still retained for some distance all round the fixation point, or whether the line of demarcation between the two divisions of the field of vision passes through this point. We should further determine the degree of blindness in the affected portion of the retina, ascertaining whether there is absolute amaurosis, or whether a feeble luminous sensation can still be aroused by a strong light; or, if the patient is unable to give this information, whether a reflex can be evoked by the approach of a lighted taper or by other means. We should also inquire whether the affected field of vision appears to the patient like a dark or grey surface, or whether it is an absolute blank of which he is as unconscious as of the existence of the blind spot. Investigation of these points is likely to throw light on the localisation of the lesion that produces hemianopsia. For example, we sometimes have hemianopsia accompanying the apoplectic or convulsive seizures of general paralysis, and as the lesion in these cases is regarded as cerebral, we should expect an amblyopic rather than an amaurotic condition of the affected field of vision. And yet in several of these cases that have come under our notice there was absolute blindness in the affected part of the retina; no objects were seen, a lighted match thrust up the eye had no effect. Wilbrand cites some interesting cases of recovery from hemianopsia with gradual appearance of the visual reflexes, but for fuller information on this subject we must refer the reader to his work.¹ Before dismissing this subject of the visual field, we may add that partial hemianopsia or sector-shaped defects of the field should be looked for, and also central and peripheral defects.

Another visual affection in which the power of fixing and following objects is lost, is *amblyopia*. Amblyopia, or dulness of vision, is a somewhat vague and unsatisfactory term. Its essential feature is diminution of the acuity of vision, with absence of such ocular defect as would account for it. In

¹ Wilbrand, 'Hemianopsie.' Berlin, 1881.

amblyopia, objects may have to be brought up to the eye to be seen, small objects may not be seen at all, colours may be misty and indistinguishable, and objects appear pale and colourless. It is evident that in such a state a patient will be very apt to make mistakes; he may easily mistake a sovereign for a shilling, and may fail to recognise many objects that are placed before him. It is then of fundamental importance in the method of examination we are pursuing that this question of the existence of amblyopia should be settled in every case, for the absence of the higher reflexes that we shall soon consider may depend on the simple fact that the objects are not distinctly seen, and not be due to any intellectual defect or defect of associations. A word of caution is necessary as to cases of unilateral amblyopia. Wilbrand is sceptical of the existence of such cases in cerebral affections, although they have been described, and he dwells on the importance in cases of this sort (*a*) of a careful perimetric examination, as hemianopsia is so easily overlooked, and (*b*) of an enquiry into the previous visual acuity of the patient, as amblyopia may have been present before, enjoining us to remember the combinations of hemianopsias, &c., that may be produced by bilateral brain affections. Amblyopia is one of the results of such combinations.

A third visual affection in which objects cannot be fixed and followed is *amaurosis*. Temporary amaurosis is a condition that deserves further enquiry. Snell¹ records a case of amaurosis fugax lasting two days; and Panas² a case of complete bilateral blindness lasting six hours. In the cerebral hemianæsthesia of Türk there is sometimes a temporary amaurosis; and in cases described by Fürstner there was unilateral amaurosis.

(3.) *Objects seen and handled*.—In this group are included the reflexes that connect vision with the simpler movements of the hand. An object is held before the eyes, and it is grasped at; if placed on a table, it is seen and picked up; if the hand is offered, it is seized. For the simplest reflex of this kind there is required, (*a*) sight of the object, as in the previous section,

¹ See 'Ophthalmic Review,' vol. i. p. 399.

² 'Gazette des Hôpitaux,' 1881.

(*b*) an idea of its position and distance, and (*c*) an idea of moving the muscles of the arm so as to reach the object. There may be no idea of what the object is; anything would be handled.

Loss of this reflex would be shown by the fact, that though objects are seen and followed with the eyes, they are not grasped by the hands. Before we can conclude such loss, however, we must remember the cautions already given, as to the state of the attention, presence of stupor, etc. As instances of disorder of this reflex, we may mention those cases in which grown-up idiots grasp at objects beyond their reach; a patient may grasp first at one side of an object then at the other, before succeeding in the attempt to seize it. Carpenter¹ cites an interesting case of a child three years old operated on for congenital cataract; who clearly recognised direction, but had little idea of distance; when told to lay hold of a watch, he groped at it like an infant lying in its cradle. These disorders are distinct from the clumsy grasping movements that depend on impairment of the kinaesthetic sense.

(4.) *Objects associated with pain recognised and avoided.*—This group implies an advance on the foregoing, in that certain sights are associated with painful experiences, and the movements are regulated by the memory of these experiences. Thus, in grasping objects, it is discovered that fire burns, an object thrust up to the eye is found sometimes to hit it; a sharp object is felt to cut. And in future an object suddenly approaching the eye causes closure of the lids, aversion of the head, protective movements of the hands; a lighted match is no longer seized; the sharp edge of a knife is shunned.

If the sight of these objects does not recall the associated idea of pain, we find an absence of these movements of avoidance. It is easy to quote instances of this. A red-hot poker may be brought up to the eye, and the patient gazes at it quite easy-minded and unapprehensive; he may even attempt to take hold of it. If he sees a lighted match blown out, he may attempt to put the glowing end into his mouth. Pretended blows cause no blinking.

(5.) *Food recognised and eaten.*—In this group we have a still

¹ 'Mental Physiology,' p. 181.

further advance. Among objects that are seen, some are recognised as good for eating, and they are seized and eaten—by the hands in the more primitive condition, with the aid of knife and fork, etc., in the more civilised. The sight of the object recalls the old associations formed by eating it, the associations of its taste, touch, etc. If these associations are lost, various curious phenomena present themselves. A person may not recognise food by sight, and never take it so long as it is simply held before him, being in fact in a condition no better than Flourens' pigeons, which would have died of hunger with food before them: but he will recognise it immediately it touches his lips, and devour it with delight. Or he may forget, and conceive some things to be food that are not; he may bite a watch, a flower, a key; he may be unable to distinguish soap from bread till he has tasted it; he may eat paper and drink ink; some bite everything held before them.

Some idiots and demented are not able to feed themselves, though they follow the spoon with interested eyes. Other patients are only able to feed themselves with their fingers, the sight of a knife and fork not suggesting the way to use them, or they may bungle in the use of them; they may eat soup with a fork, or try to cut with the back of their knife, or drink from their bread, and raise the saucer to their lips and bite it.

We have been considering only the commonest kinds of food and instruments of eating, such as are in daily use. If the associations are less organised, if, for example, a glass of acid were swallowed for a glass of water, or a poison-berry were taken in mistake for a hip, the act falls into a higher category.

(6.) *The Visual Reflexes in walking and the allied movements.*—In a healthy adult sight contributes little to those guiding sensations which play such an all-important part in maintaining the balance of the body; the sensations of sight are only seriously called on when the kinæsthetic sense is impaired. Sight, however, is in constant requisition in locomotion. It enables us to walk in a straight line, avoid obstacles, go up steps readily, sit down safely, etc. When the visual associations that enter into these movements are absent or impaired,

we find the patient stumbling against obstacles, jostling against people, running against lamp-posts, doors, or walls. In one of Reinhard's cases the patient ran against a door and stumbled against the furniture in her room, sometimes even though it was staring her in the face. She seemed incapable of judging distance, and did not know when she came up to an object, often the first intimation of the fact was her knocking against it. When she did notice an obstacle she gave it an absurdly wide offing.

Obstacles are seen, but they no longer suggest the movements that are required to avoid them. Past experience, in other words old associations, are not available, or, at all events, are not made use of. The more unusual the obstacle, the less likely is the patient to avoid it. Though able to walk about with a degree of safety at home, he may be quite unable to steer his way in an unknown house, or on uneven ground. Sight of a train noiselessly approaching, or of an open grating in a street, may suggest no danger, and cause no alteration in his course. The impairment of this reflex is also seen in a person's attempting to get on a chair, or jump down from one. There seems a want of association between the judgment of the distance involved and the movements to attain this distance.

(7.) *Objects recognised and used properly.*—In this group the test of recognition is the ability to use properly. In applying the test it is important to eliminate the complications that arise from the association of sight with the other senses, especially with touch. For example, a key is held before a patient; if he points to a door and makes a turning movement with his hand, it is clear that the mere sight of it has suggested its use. But if the key has to be put into his hand, and he has to examine it with his fingers before he knows what it is, then we have to deal not with sight alone, but with sight associated with touch. A good example of this is seen in a case of Critchett's, cited by Carpenter:¹ A young woman, blind from birth, whose sight had been imparted by an operation, was not able to ascertain what an object was by sight alone; she would describe the shape, colour, glistening appearance of

¹ Loc. cit.

a pair of scissors, but could not tell what they were till she had put her fingers in them. Here the sensations of touch contributed more than those of sight in determining the use of an object.

The sight of an object calls up ideas of weight, touch, smell, taste, sound, and it is by these associations that an object is recognised, and its proper use rendered possible. When these associations are destroyed, the power of recognition and use is lost, and we have one of the conditions to which Zussmaul applies the term "apraxia." Instances of this condition have already been referred to under former heads; we shall now give others that occur in connection with objects a degree less simple.

Objects of everyday use, such as a watch, penknife, book, pipe, thimble, umbrella, etc., are looked at vacantly, and no signs are made that betoken recognition. Even if placed in the patient's hand they are handled strangely, or played with in an idiotic manner; the watch is not put up to the ear, or the knife opened, or the pipe put into the mouth. A match-box with matches in it is turned round and round with an air of helpless ignorance. In one case a patient knew what his bed, a piece of bread, and a fire were, but not what a needle, a shoe, or his clothes were. He may have forgotten the simplest properties of things, and may busy himself trying to bend a piece of wood or iron; indeed, a person in this condition would no doubt, if opportunity offered, good-naturedly stroke a tiger, or attempt to coax a lion. An object may be recognised as a member of a class, but not as an individual. In the higher instances of individual recognition, call is made on finer and more numerous associations, and the act is removed from the group of comparatively simple reflexes at present under consideration. Our knowledge of an object is very much a matter of the associations it arouses.

The recognition of well-known faces, though scarcely falling within the literal heading of this group, may be conveniently treated of here. The patient may have no idea that the people who wait on him are his wife and children; an old friend is not greeted in the accustomed way; a stranger is taken for an old acquaintance. Ribot mentions a case where the physician

was not recognised, though he came every day for fourteen months; and a patient recently told me that his wife and child, who had been visiting him, were good imitations, and well made up, but he was certain they were nothing more—for one thing, the child was not large enough.

It may be that objects are still recognised, but only dimly and with uncertainty; they appear new and strange. For example, a patient's hands may look odd and queer to him, quite different from what they used to look; and familiar places and buildings may seem so strange, that he will doubt whether he has seen them before.

(8.) *The Visual Reflexes in Dressing, etc.*—Dressing is one of our acquired semi-automatic acts, and it requires that particular articles should be recognised, and their use and the order in which they should be put on known. Sometimes we find that the articles of dress are not recognised by sight; a shoe, a hat, a shawl, may be staring a person in the face, and yet suggest no ideas as to their use. He may have forgotten how to put his clothes on; he may spend an hour fumbling at his trousers, and at the end be no nearer the goal than when he started; he may put his waistcoat over his coat, or look aggrieved when a fourth coat in which he is just preparing to robe himself is taken from him. With his eyes wide open he may persist in trying to thrust things into a supposed pocket, though a moment's glance should convince him that there is no pocket there. He may, like a celebrated Scotch professor, go about with his wife's white stocking on one leg, and his own black one on the other, and be continually doing similar acts. He may lie down on a chest of drawers, and think it is a sofa.

(9.) *The Visual Reflexes in Handicrafts and Arts.*—The reflexes embraced in this section are on a distinctly higher level than those previously considered, and it is correspondingly difficult to distinguish the part that sight bears in them. Still, sight and its associations enter largely into such actions as sewing, drawing, reading music at sight, boxing, digging, and the rudimentary operations of most manual employments; and it is of importance to test a patient in respect to actions of this kind to which he has been accustomed. If he is unable to

perform them, bungling simple work, and failing in what used to cost him scarcely an effort, we have reason to conclude, due regard being paid to other disturbing influences, that the visual reflexes are impaired, and we have a valuable indication as to the state of the patient's brain. As an example of disturbance under this head, we may mention the case of a man who used to sketch well, but who after a cerebral attack would draw a man's head that a child might be ashamed of. Other examples will readily suggest themselves to the reader.

(10.) *Gestures understood*.—Pantomime is the simplest form of language, and is almost universal in its application; it falls therefore to be considered before the higher reflexes that are concerned in naming objects and in reading. We should ascertain whether the patient acts properly when a hand is held out to him. Does he protrude his tongue when we point to his mouth, or go so far as show him our own? Does he come to us if we beckon him, or does he look in the direction we point? Will he produce his knife when we go through the pantomime of sharpening a pencil, and point to his pocket? It is unnecessary for us to say more on this head, the absence or impairment of the power of understanding simple gestures betrays affection of certain visual reflexes.

(11.) *Objects named*.—From our earliest childhood we are accustomed to associate the sight of many familiar persons and things with their names. When this association fails, and the sight of the object does not recall its name, it is only another way of expressing the same fact to say that a visual reflex is in fault. In such a case, we have to inquire what class of objects cannot be named; whether those that we only rarely see and name, or those that we see and name almost every day of our lives; thus a man may have forgotten the names, bread, water, grass, or may not remember his wife's name. In other cases the power of naming objects may not be wholly lost; the patient may give some name closely allied, or may be able to find the name of the genus though not of the species, or may enter into a description of the article and seem to know everything about it but its name. Again, sight alone may fail to suggest the name, but sight, aided by some other sense as touch, hearing, etc., may recall it. The subdivisions of this

group and the different points to observe are too well known to need further notice here.

(12.) *Objects counted and figures read.*—It should be seen whether the patient can tell or indicate the number of fingers shown him, or can count correctly a series of objects laid out before him. We may find that the patient is able to count a number of large objects, but is unable to count small objects that are placed close together; for example, a string of beads. The presence of amblyopia would explain this; but there seem to be cases where the individual objects are distinctly enough seen and yet a series cannot be followed, owing either to some fault of nervous conduction, or to some central defect, probably a kind of irradiation.

We should also determine whether the patient is able to read figures. Can he tell individual figures, and combinations of them? 2758, for example, may be read 2700, 50, 80; and there are many varieties into which we need not enter. Can he tell the time on a watch? Can he do simple addition? Mlle. Skwortzoff¹ cites a case in which the power of reading figures was retained, while that of reading letters was lost.

It should also be noticed whether the value of the numbers is appreciated, or whether it is merely the names that are remembered. An estimate of this can be formed by the appearance the individual makes in a game at dominoes or cards.

(13.) *The Visual Reflexes in Reading.*—A person may see a page of print, know that it is print, be able to tell when it is upside down, notice the paragraphs, italics, capitals, and even be able to find a given place again, and yet be quite unable to read; just as he may recognise handwriting, or know his own name without being able to decipher a letter. This is the affection known as word-blindness.

A step higher, and he is able to name individual letters, or syllables, or words. He may be able to read some words and not others. He may mispronounce letters and words, or give altogether wrong names to them. He may skip words in reading, or pass over lines, or go from the middle of one line to the next. He may read in this way, and have scarcely an idea of the meaning of what he reads.

¹ Mlle. Skwortzoff, 'De la Cécité et de la Surdité des Mots.' Paris, 1881.

Still a step higher, and he can not only read, but can understand what he reads. Individuals who have lost this power remind us, says Mlle. Skwortzoff, of persons who remember the rules of pronunciation of a foreign language, and are able to read it without understanding a word of it. Short simple sentences may be comprehended, while complicated ones only bewilder. If the patient is aphasic, we can test his ability to understand what he reads by writing down some simple request and observing whether he complies with it. It is not sufficient to get him to turn over the page while we are reading, or point out the passage read, for we must remember that a patient may recognise a word when it is uttered and yet be unable to read it without this help.

(14.) *The Visual Reflexes in Writing.*—We must distinguish between ability to copy writing, and ability to write spontaneously or from dictation. Copying is in some cases merely an instance of drawing; indeed the patient may have so forgotten the once almost automatic art of writing, that he may begin at the last letter of a word instead of the first. We should examine every patient and see whether he is able to copy written or printed text, and can convert the latter into written characters.

Writing spontaneously or from dictation is a complicated process which it is not easy to analyse satisfactorily. Many educated people can write with their eyes closed, and many cases have been reported of patients being unable to read what they had just written. As we are now dealing with sight only so far as it is the starting-point of a reflex, we must limit ourselves to the consideration of the influence sight exercises on the act of writing. Under the influence of sight the letters are kept at proper distances from one another, an approximation to a straight line is maintained, and glaring errors of orthography or composition are detected. In some brain-diseases, the reverse of all this is observed; for example, the letters are badly formed, or heaped one on the top of the other, or the lines may not be kept to, or a commencement is made in the absurdest places, or striking errors pass unnoticed. Examination of the writing of a patient should not be omitted where an inquiry is being made into the condition of sight.

(15.) *Higher Visual Reflexes*.—We are now arrived at the more complex and less organised reflexes which it is difficult to group together satisfactorily, and we shall be content with the easier plan of pointing out the steep and thorny way of investigation instead of treading it ourselves. Are the more complex and intricate visual presentations understood by the patient as they once were? Can he remember his way about in his own neighbourhood or in less familiar districts? Does he grasp the significance of what he sees happening around him? If, for example, he saw two men fighting desperately, would he recognise the fact, or merely stare at them, or think they were amusing themselves? Does he appreciate changes of facial expression and demeanour? Is he as alive to beauty of form or colouring? Does he understand symbols and symbolic actions? In other words, are the same delicate associations called up by the sight of objects as formerly were? These higher visual reflexes carry us into the highest reaches of the mind, and their analysis and due appreciation naturally present difficulties that are great even where normal brain-action is under consideration. How much greater are they when the problem is overlaid by the vast disturbing influences of disordered brain-action and its offspring, protean Insanity! But are we entitled to conclude that sight is unimpaired until these higher regions of intellectual sight have been explored? For our part we think not, and we would not hesitate to say that if a Wordsworth became as a Peter Bell, no longer feeling the witchery of the soft blue sky, regarding a primrose by a river's brim as but a yellow primrose and nothing more, he would in our opinion be suffering from an undoubted visual defect.

We have confined our attention chiefly to the intellectual side of vision, but in a complete view of the subject regard would also be had to the manifestations of the emotions that are called forth by sight.

To be continued.

AN ANALYSIS OF THE NERVE-PHENOMENA IN A CASE OF ANÆSTHETIC LEPROSY.

BY W. ALLEN STURGE, M.D.

MR. D. aged 53, consulted Mr. Hutchinson in July 1881, through whose kindness I was enabled to see the patient. The patient said that as a young man he had good health until the age of 23, when he went to reside in Bermuda. There he had a severe illness, which he believes was a malarial fever of some kind. Shortly after his recovery from this illness he began to suffer from a skin-disease, which appeared first on the front of the abdomen, and gradually spread to the back, arms and legs; but, so far as he can recollect, did not affect the hands and feet. This eruption lasted for many years, and then gradually subsided. At the present time there are a few patches on the back, and on the right arm, which are not distinguishable from ordinary psoriasis. The patient states, however, that he was seen by Dr. Brown-Séquard when the eruption was at its height, and that he pronounced it to be the eruption of leprosy.

Not long after the onset of the eruption he began to suffer from hyperæsthesia of the extremities. The feet became so tended that it inconvenienced him to stand upon them, and it hurt him to clench things tightly in the hands. The hyperæsthesia gradually extended up the limbs. As time went on, this hyperæsthesia became less and less marked, until at last it was replaced by numbness, at first slight, but gradually increasing until it became complete. The onset of anæsthesia was accompanied by the complete cessation of the sweat function in the parts of skin affected by it.

As the modification in sensation has progressed, he has gradually noticed that his limbs were getting weak, and that some of the muscles were wasting.

At the present time there is complete anæsthesia over the whole extent of the upper and lower limbs, and over areas of the trunk adjacent to the limbs. The greater portion of the trunk is, however, free from it. Between an anæsthetic part of the skin and the adjacent healthy portions there is a border of skin, from half-an-inch to an inch wide, where there is a moderate degree of hyperæsthesia, and these borders obviously point to the fact that the disease is still slowly advancing. Wherever the skin is anæsthetic it is thin, dry, more or less shiny, and the sweat-glands and hair-follicles appear to be completely atrophied.

There is complete atrophy of all the small muscles of both hands, which have assumed the most marked "main-en-griffe" position. In the left arm there is complete wasting of all the muscles on the back of the forearm, including the supinator longus; and there is a slight degree of wasting in the muscles of the front of the forearm. The biceps, brachialis anticus and triceps are nearly or quite healthy, but the deltoid is markedly wasted. In the right arm there is no wasting of the muscles on the back of the forearm; a slight degree of wasting of the muscles on the front of the forearm; none of the biceps, brachialis anticus, or triceps; but marked wasting of the deltoid. The other muscles about the shoulder-joint and the serratus magnus were healthy on both sides. There was wasting of the small muscles of the feet; complete wasting of the muscles on the front of both legs; but little, if any, of those of the calves or of the thighs.

The muscles of the back, chest and abdomen were healthy. The face presented a curious condition; there was complete anæsthesia of the forehead and eyelids, but the rest of the face and the scalp were ordinarily sensitive to touch. There was a corresponding atrophy of all the muscles beneath the anæsthetic skin, viz. the occipito-frontalis, the corrugatores supercilii and the orbiculares palpebrarum. Some years ago he began to experience difficulty in shutting his eyes, which increased, until all power of moving the eyelids had gone. From that time he began to suffer from severe inflammation in the eyes, and at last, after many remedies had been tried and one or two operations had been performed, he lost his

sight altogether, and to prevent irritation the eyelids were sewn together. The muscles of the rest of the face are healthy, as are those of the tongue, throat and larynx, in proof of which he states that his voice is exceptionally strong, and that he has no difficulty in making himself heard clearly at large public meetings.

It is a point of much interest that, although the skin of the limbs is so completely anæsthetic, there is apparently little or no anæsthesia of the deeper structures. He is able to feel *deep* pressure quite easily, and he even feels a pinch quite well, if other structures than the skin be involved. I regret that I did not observe whether this sensitiveness of the deeper structures was also present in those places where the muscles were extensively atrophied. For reasons that I shall presently state, I think it would be important in any similar case to note this.

His brain power throughout has been excellent, and, notwithstanding his crippled condition, he is a distinguished member of one of the learned professions.

The diagnosis in this case was that it was an old-standing case of leprosy in which the anæsthetic symptoms were exceedingly well marked, whilst the skin symptoms were only moderately so; in other words, that it was a case of non-tuberculated anæsthetic leprosy. I shall endeavour to make an analysis of the phenomena present, with a view of throwing further light, if possible, upon the pathology of the nerve-phenomena of the disease. I regret that in the single, somewhat hurried interview that I had with the patient I omitted to note several points of importance for this purpose; but I think we have sufficient data to enable us to arrive at definite conclusions in regard to some of the questions which may arise.

We have to consider, (1) the primary seat of the nerve lesion; (2) the course followed by it in its progressive advance.

The primary seat of the nerve lesion.—The questions which present themselves in this connection are: (1) Is the lesion *central*, i.e. in the brain or spinal cord? or is it *peripheral*, i.e. in the nerves? (2) If peripheral, is it (*a*) in the trunks of the

nerves? (*b*) in the finer nerve branches? or (*c*) in the peripheral terminations of the nerves?

Answers to these questions may be sought for by an examination—(1) of the sensory phenomena, (2) of the motor phenomena.

1. *The sensory phenomena.*—The patient's description of his sensory symptoms was that he first noticed hyperæsthesia of the extremities; the feet were tender, and it was painful to hold things firmly in the hands. The hyperæsthesia gradually extended up the limbs, and after a time it slowly diminished, being replaced by anæsthesia, which ultimately became complete. On examination, we found that all four limbs were completely anæsthetic, together with a strip of skin in the adjacent portions of the trunk. The anæsthetic areas were still bordered by a band of hyperæsthesia.

The first thing to observe about this account is that the mode of invasion of the anæsthesia is wholly unlike anything that is met with in central disease. The patient was quite clear in his statement that it began at the extremities of the limbs, and gradually crept up towards the trunk, and this statement is borne out by the border of hyperæsthesia still existing. It is also quite unlike the history we should get if the nerve-trunks themselves had been primarily involved. In that case we should have had the anæsthetic patches taking shapes corresponding to the distribution of sensory nerves. It may be said that we have only the patient's own statement that the anæsthesia did not come on in this way, and that he would be unlikely to notice the exact mode of onset of his numbness. Against this I would urge that he was a man of high and cultivated intelligence, that the anæsthesia has been gradually advancing for more than twenty years, and that he has carefully watched his own symptoms during that time. The present margin marking the advance of the disease shows no sign whatever of following the lines of certain nerve distributions.

Thus then we are driven to the conclusion, that the primary seat of the nerve lesion is not in the central nervous system, nor in the trunks of the nerves. It must therefore have been in the ultimate branches of the sensory nerves, or in their peripheral terminations.

Another point in favour of the conclusion, that neither the central organs nor the trunks of the nerves were primarily affected, is the fact to which I drew attention, that although the skin was absolutely anæsthetic, yet he was able to feel deep pressure quite easily, and he even felt a pinch quite well if other structures than the skin were involved. Unfortunately I have no note of the relative deep sensibility of parts where the muscles were healthy, as compared with those where they were atrophied. My conviction is that there was no sensation in the atrophied muscles. In anæsthesia from central disease, no such condition as this is observed. A limb is wholly or partially anæsthetic in its entirety; or if, as sometimes happens in locomotor ataxy, a district of a limb becomes anæsthetic, we have no such superficial anæsthesia as that here described.

Similarly in disease of a nerve-trunk, complete or partial anæsthesia of all parts superficial and deep supplied by that nerve ensues; but here also a purely superficial anæsthesia is never met with.

Thus then all the sensory phenomena in this case point to the conclusion, that it is the peripheral terminations of the nerves that were primarily involved.

Before passing on to the next division of the subject, viz. the motor phenomena, let us see whether we can get evidence to prove that the mode of invasion above described is a usual one in cases of non-tuberculated leprosy. For this purpose I cannot do better than refer to the masterly treatise of Dr. Hillis on "Leprosy in British Guiana."

In his chapter on non-tuberculated leprosy, Dr. Hillis describes the following cases:—

(1.) A man, æt. 64, affected for 8 years (Case XXI. p. 84). There is cutaneous anæsthesia of both arms from the fingers to four inches above the elbow-joints, and from the toes to the ankle-joints inclusive.

(2.) A man, æt. 47, affected for 9 years, (Case XXVI. p. 89). He had complete cutaneous anæsthesia up to the insertion of the deltoid in the left arm, and in the right arm up to the elbow-joint in front, and to the middle of the back of the forearm. Complete cutaneous anæsthesia of the feet and legs up to the knees.

(3.) A man affected for 15 years (Case XXXIII. p. 97) is described as having lost sensation in all the limbs as far as the knees and elbows.

Now when we consider how various are the nerves which supply sensation to the skin of the limbs, and from what different sources they are derived, it will be at once evident that no affection of nerve-trunks would give rise to the distribution of anæsthesia described in these cases. I am therefore justified in quoting them as further proof of the conclusion at which I above arrived. I must however state, that Dr. Hillis believed that he could, in certain cases, trace a connection between the seat of cutaneous phenomena and the distribution of nerve-trunks. I gather, however, from his remarks, that he has more especially noticed this in the distribution of the eruption, rather than of anæsthesia, a point upon which the case I am reporting offers at the present time no evidence. Thus he says: "Although in some cases it would appear as if the patch [of eruption] corresponded to the distribution of a nerve, such is not always the case. I have, however, been struck with how frequently the course of the musculo-spiral nerve is apparently taken by this eruption" (p. 74). Of two cases, however, where the eruption was supposed to take this course, I find that the report is too vague to justify us in laying much stress on this point; and when we consider the scattered and serpiginous nature of the patches of eruption, it would not be surprising that some of the patches might appear to follow the course of some one nerve. But, however it may be with the eruption, in no case that he describes did the anæsthesia correspond to the distribution of one nerve; where it was not simply patchy and thoroughly irregular, it in nearly every instance assumed the form described in the case above reported, and in those quoted from his own book; viz. a gradual creeping up the limbs, without reference to the nerve supply.

I now come to the next branch of the subject, viz.:

2. *The motor phenomena.*—The mode of onset of the muscular atrophy in the limbs differed widely from that of the anæsthesia; for whereas we saw that the latter advanced without reference to the geographical distribution of nerves, the

muscles were atrophied by groups, certain groups being intact, whilst other groups in their near neighbourhood, and placed very similarly as regards distance from the centre and in relation to the anæsthesia, were completely atrophied. This distribution of the atrophy points to the conclusion, that the nerve lesion giving rise to the atrophy was situated either in the spinal cord or in the trunks of the nerves. At first sight, it may not appear to be an easy thing to decide whether a given muscular atrophy is due to a central (spinal cord) lesion, or to disease of the trunks of the nerves. There is, however, one infallible test which will, I believe, be found never to fail, and which is not generally known. I refer to the distribution of the atrophy, which is quite different in the two cases. When the trunk of a nerve is affected in such a way as to produce atrophy, all the muscles supplied by the nerve below the seat of lesion will be equally involved—muscles belonging to different groups, and having little or nothing in common, beyond the fact that they are supplied by the same nerve. In the case of spinal-cord disease, those muscles will tend to be simultaneously affected which are associated together in the performance of certain co-ordinated movements, whether they be supplied by the same nerve or not. This is a statement which can, I believe, be confirmed by a careful analysis of the distribution of the atrophy in any case of muscular atrophy where the distribution is partial. It was clearly proved by two cases, which formed the basis of a paper read by me before the Medical Society of London.¹ In any given case of atrophy of a limb, the muscular grouping according to nerve supply should be carefully thought out and compared with the grouping of the atrophied muscles. In the case of the arm, this is done with great ease, for there is a well-marked group of muscles co-ordinated for a single purpose, viz. to bend the elbow, which does not correspond to the grouping, according to nerve-supply. Three muscles take part in this action—the biceps, the brachialis anticus, and the supinator longus; the two former being supplied by the musculo-cutaneous nerve, and the latter by the musculo-spiral. Disease of the musculo-cutaneous nerve will therefore cause

¹ 'Proceedings of the Medical Society,' vol. v. p. 357.

atrophy of the biceps and brachialis anticus; without the supinator longus; whilst disease of the musculo-spiral will cause atrophy of the supinator longus, without the biceps and brachialis anticus. Now it has been shown, on the other hand, that disease of the spinal cord giving rise to atrophy of any one of these muscles gives rise to atrophy of both the others also; whilst in disease of the spinal cord giving rise to atrophy of the muscles of the back of the forearm (which are supplied by the musculo-spiral nerve), the supinator longus, which is also supplied by that nerve, will not be atrophied unless the biceps and brachialis anticus are also involved.

It fortunately happened that in the left arm of our patient the exact condition was present which is required to enable us to make use of this criterion; for the muscles of the back of the forearm were completely atrophied, whereas the biceps and brachialis anticus were intact.

What was the state of the supinator longus? It was completely atrophied. Hence we may conclude, without fear of error, that the lesion giving rise to the muscular atrophy is situated in the trunks of the nerves and not in the spinal cord.

Thus we are brought to the conclusion, that the affection of the sensory nerves is independent of the distribution of the nerve-trunks, and that the primary lesion giving rise to it is situated in the peripheral terminations of the sensory nerves; whereas the motor affection corresponds with the distribution of the nerve-trunks, and that the lesion giving rise to it is seated in those nerve-trunks. The questions arise:

Is there any connection between these two diversely located lesions? and can any explanation be found for this curious mode of election of the disease?

After carefully considering these questions, I believe that the following is the true explanation of the phenomena. It is evident that the morbid process which gives rise to the leprous condition has a strong tendency, in the non-tuberculated variety, at any rate, to advance by continuity of tissue. Dr. Hillis, in summing up this variety of eruption, says: "At first small, the spots subsequently enlarge, and may by coalescing take over the greater part of the body. . . . they

have a tendency to spread serpigiously and peripherally.”¹ It is evident that in this form of the disease it is the skin and nerves which have a special tendency to be attacked; and therefore we might expect to see the same tendency to advance by continuity of tissue in the case of the nerves, as in the case of the skin. I have given reasons for believing that the nervous affection begins at the peripheral extremities of the cutaneous nerves. From this point the disease will creep up the nerve-fibres, and from individual nerve-fibres it will extend upwards to the point where several fibres are collected together into a small nerve-trunk, and up this to the larger branches, and eventually to the main nerve-trunk. The nerve disease is proved by microscopical examination to consist mainly of a cellular new formation, accompanied by some hypertrophy of the connective tissue binding the fibres together. The result of this is that those fibres of the nerve which have not already been injured by the new growth become squeezed, and ultimately destroyed. The branches given off by a nerve-trunk to the muscles supplied by it are, for the most part, independent of the cutaneous branches of the same nerve. As the disease begins in the peripheral extremities of these latter nerves, the motor nerves will not be affected until it has passed up the cutaneous branches to the main branch or main trunk from which the motor nerves are given off. When this has happened, a whole group of muscles will be affected simultaneously—a group deriving its nerve-supply from a common origin.

Let us see whether the distribution of the muscular symptoms in Mr. D.’s case offers any support to this view. To do this, it will be necessary to recall the relations which are borne to one another by the sensory and motor branches of the various nerves of the limbs. In making this review, there are three points to which attention must be drawn in connection with each nerve.

(1.) The position in the limb occupied by the area of skin supplied by the nerve, *i.e.* whether nearer the trunk, or farther from the trunk. Bearing in mind the mode of invasion from the peripheral extremity of the limb towards the centre, it is

¹ Loc. cit., p. 101.

evident that areas of skin further from the trunk will have become affected sooner than those nearer the trunk, and hence that, if my explanation be the correct one, the disease will have had a longer time to travel up the nerves ascending from the areas more distant from the trunk than up those passing from the less distant areas.

(2.) The distance from the peripheral extremity of the sensory nerve to the nearest point where it meets with motor nerves. This is obviously a point of great importance, for if a sensory nerve which may have become affected very early in the disease was given off from the nerve-trunk high up in the limb, as is the case, for instance, with the internal saphenous nerve, the distance to be travelled by the disease would be so great that it might take very much longer to reach the trunk of the nerve, or the motor branches, than in the case of a nerve affected much later in the disease, but offering relatively a much shorter course before reaching motor fibres.

(3.) The size of the sensory nerve both absolutely and relatively to the nerve-trunk or branch from which it springs. A large or widely branching sensory nerve, such, for instance, as the circumflex, might be expected to offer an easier passage for disease than a single small sensory branch of equal length; and the disease would, in all probability, reach the nerve-trunk in a shorter time in the case of the former than in the case of the latter. Whether this be so or not, however, it is certain that disease passing up a sensory nerve of considerable size, and reaching a motor nerve of about the same size, will produce a more rapid and intense effect than if it travels up a sensory nerve of small size, and reaches a nerve-trunk of relatively much greater size. Compare, for instance, disease travelling up the radial nerve to meet the posterior interosseous, and disease passing up the internal cutaneous of the musculo-spiral to meet the main trunk of the musculo-spiral nerve.

Each of these three points must be borne in mind in considering the various nerves. I will begin with

I. *The Nerves of the Arm.*—Here we have five main nerves:

(1.) The musculo-spiral.

(2.) The musculo-cutaneous.

- (3.) The median.
- (4.) The ulnar.
- (5.) The circumflex.

(1.) *The Musculo-spiral*.—The *motor* branches are :

- (a) Branches to the triceps, given off high up in the course of the nerve.
- (b) Branches to the supinator longus and extensor carpi radialis longior, given off after passing through the external intermuscular septum, and before the nerve divides into the radial and post. interosseous nerves.
- (c) The *posterior interosseous* supplying the supinator brevis, the extensor carpi radialis brevior, and all the extensor muscles at the back of the forearm. The posterior interosseous splits off from the radial just below the elbow-joint.

The *sensory* branches are :

- (a) The *internal cutaneous of the musculo-spiral*, a small nerve which takes its origin high up in the course of the nerve, and passes to supply the skin of the inner and upper part of the arm. Applying the three tests above mentioned, we see that the area of skin supplied by the nerve will be attacked very late in the disease ; the disease, however, will not have a great distance to travel up the nerve before reaching the main trunk ; and thirdly, that the sensory nerve bears a very small proportion to the main trunk from which it issues.
- (b) The *two external cutaneous branches*.—These which, taken together, make a moderate-sized nerve, have their origin just after the musculo-spiral has pierced the external intermuscular septum, and before it has given its branches to the supinator longus and ext. carpi rad. longior. They are distributed, the upper to the skin of the lower half of the upper arm in its anterior aspect ; and the lower to the skin above and in front of the elbow-joint, and to the outer part of the upper half of the forearm. The

area supplied by them will therefore be affected about the middle period of the disease ; the disease will have, on the average, a moderately short distance to travel to reach the trunk of the nerve ; and the proportion borne jointly by the nerves to the main trunk, though not approaching equality, is a considerable one.

- (e) The *radial nerve*.—This is a nerve of considerable size, taking its origin at the bifurcation of the musculo-spiral nerve just below the elbow-joint, and running a long course to supply the skin of a portion of the hand. The area of skin supplied by it would therefore be one of the first to be attacked ; but the disease would have a considerable distance to travel before meeting with a motor branch ; the proportion borne by the sensory nerve to the motor branches from which it split off is, roughly speaking, one of equality.

If then my explanation of the course of the disease be the true one, how would the muscles supplied by the musculo-spiral nerve be likely to be affected ? None of the sensory branches are so placed as to make muscular involvement particularly easy, and there is no nerve which is both affected early in the disease and which offers a short course. The radial, however, is affected early and completely ; it is a relatively large nerve, and offers a considerable pathway for the disease, which will therefore be likely, after an interval of some length, to reach the motor branches, and thus to cause atrophy of the muscles at the back of the forearm. The external cutaneous nerves will be affected much later, but offer a much shorter course to the disease, which, roughly speaking, might be expected to reach the trunk of the nerve at about the same time as by the radial, and thus to cause atrophy of the supinator longus and ext. carpi radialis longior. The internal cutaneous of the musculo-spiral will however be affected so late in the disease, and it is so small in relation to the main trunk from which it comes, that it is highly unlikely that any atrophy of the triceps would take place.

(2.) *The Musculo-cutaneous nerve.*—The motor branches of this nerve come off in the upper arm—for the most part in its upper half—to supply the biceps, the coraco-brachialis and the brachialis anticus.

The sensory branches supply the skin of the outer part of the forearm, chiefly in its lower half. The area of skin supplied by it will hence be affected some considerable time after the first onset of the malady, and the distance to be travelled by the disease is very great, the muscular branches taking their rise from a part of the nerve that is far removed from the peripheral cutaneous distribution. Hence it is highly unlikely that any atrophy of the biceps coraco-brachialis and brachialis anticus would be met with.

(3.) *The Median.*—The motor branches are :

- (a) Branches given off just below the elbow to the flexor sublimis digitorum, part of the flexor profundus, the pronator radii teres, the flexor longus pollicis, the palmaris longus, and the pronator quadratus.
- (b) Branches given off below the wrist to some of the small muscles of the hand.

The sensory branches are confined to the supply of part of the hand.

Thus then the area of skin supplied by it will be affected at the very onset of the disease; the nerve up which the disease will travel is one of considerable size; the motor nerves to the small muscles of the hand will be quickly met with, whilst those to the group of muscles on the front of the forearm will be reached very much later. We may therefore expect that the small muscles of the hand will be quickly affected. As regards the others, we may observe that the relation borne by the motor branches to the sensory trunk of the nerve is strictly comparable to that borne by the radial nerve to the posterior interosseous, the areas of skin supplied by the two sensory nerves being situated in precisely analogous positions as regards the first onset of disease; and the branching off of the motor nerves being at almost precisely the same distance up the limb in the one case as in the other. Thus we may conclude, that the chances in favour of the posterior group of muscles being affected are practically equal to those

of affection of the front group of muscles. In a given number of similar cases of disease we might therefore expect that sometimes one group of muscles, and sometimes the other, would be first to be affected, a slight variation in priority of attack in the back or the front of the hand being sufficient to produce this variation in the subsequent motor affection. There is this further remark to be made, that whereas in the case of the median, disease of the upper part of the nerve can only have advanced from the hand, in the case of the musculo-spiral the corresponding advance of disease from the hand will be augmented by disease (beginning later, but having less distance to travel) advancing up the two external cutaneous nerves. *Ceteris paribus*, therefore, we should expect a more intense degree of motor affection in the case of the musculo-spiral than of the median.

(4.) *The Ulnar nerve*.—The *motor* branches are :

- (a) Branches to the flexor carpi ulnaris, and half the flexor profundus digitorum. These arise from the trunk of the nerve just below the elbow.
- (b) Branches coming off below the wrist to supply certain of the small muscles of the hand.

The *sensory* branches are :

- (a) Branches rising from the nerve in its course down the forearm, to supply the skin of the inner side of the lower half of the forearm.
- (b) Branches to the skin of portions of the hand.

What we have to say here is very much a repetition of what was said of the median nerve. The nerves to the skin of the hand will be affected at, or soon after, the onset of the disease, which having but a short distance to traverse before meeting with the motor nerves to the small muscles of the hand, will quickly produce atrophy of these muscles. From this point it would take about the same time to reach the nerves of the large muscles of the forearm, as in the case of the median and radial nerves ; only that, in the case of the ulnar, the morbid action will be reinforced by the disease passing up from the sensory nerves above mentioned as being distributed to the forearm, which, however, will naturally have been affected

much later than those of the hand. On the whole, therefore, we might expect the upper motor branches of the ulnar to be affected a little sooner than those of the median or the posterior interosseous nerve.

(5.) The *Circumflex nerve*.—Its motor branches go to the deltoid and the teres minor muscles.

The sensory branches supply the skin over the deltoid muscle. With regard to this nerve, we may remark that the motor and sensory portions are, roughly speaking, equal in size, and are intimately related to one another in their distribution, which has not been the case, to anything like the same extent, in the other nerves that have been described. The sensory part of the nerve is short and widely branched. Disease once established in the peripheral termination of the branches, has many roads open towards the centre, and it has but a short distance to traverse before reaching the motor branches. On the other hand, the area of skin supplied by the nerve will have been one of the last to be attacked; but, when once attacked, no long time would, for the above reasons, be required to show motor involvement, which would be likely rapidly to become complete.

Treating the arm as a whole, then, we might expect that all the small muscles of the hand would be early and completely affected. The groups of muscles on the front and back of the forearm, supplied by the median and posterior interosseous nerves respectively, might or might not be involved, the chances in favour of affection of the front group being about equal to those of affection of the group at the back; but if both be affected, the muscles of the back group are likely to be more extensively atrophied than those of the front group. Moreover, if any one muscle of either of these groups be affected, all the other muscles of the same group will also be affected and probably in the same degree. For reasons above given, the chances in favour of affection of the muscles supplied by the ulnar nerve will probably be rather greater than in the case of the two groups of muscles just mentioned. The supinator longus and ext. carpi rad. longior would tend to be attacked by extension of the disease either up the main trunk of the musculo-spiral, or up the ext. cutaneous nerves, the

chances of such attack being roughly equal to, perhaps somewhat less than in, the case of the median and posterior interosseous groups. The triceps would almost certainly escape for the reasons above detailed, and the same remark will apply to the biceps, coraco-brachialis and brachialis anticus. If the latter group were attacked, the brachialis anticus might be expected to be the first to succumb. The deltoid, notwithstanding that the skin supplied by the circumflex nerve would be some of the last to be attacked, would probably rapidly become affected when once the disease had extended up to the height of the shoulder-joint.

In approaching the subject thus from the *à priori* standpoint, I do not think I have said more than the conditions postulated justify. Let us compare the picture thus drawn with that presented by the account—unfortunately only partially complete—of our patient. In him all the small muscles of both hands were completely atrophied. In the left arm the muscles of the group supplied by the posterior interosseous nerve were much atrophied, those supplied by the median being slightly atrophied. In the right arm the muscles supplied by the median were slightly atrophied, whilst those supplied by the posterior interosseous were comparatively healthy.

This distribution of atrophy in the two forearms strictly bears out what was said about the equality of the chances of these two groups of muscles becoming affected. As regards the supinator longus and extensor carpi radialis long., these were completely atrophied in the left arm. Unfortunately I have no definite note of their state in the right arm; and the same remark, I regret, applies to the muscles supplied by the ulnar nerve. I believe, however, that these were completely atrophied in the left arm, but I am unable to say anything of their condition in the right. The triceps was healthy on both sides, as were also the biceps, brachialis anticus, and coraco-brachialis; whereas, on the contrary, the deltoid was markedly atrophied on both sides.

I think that, notwithstanding the absence of a complete definite record of every muscle, which under the circumstances it was not in my power to make, the close correspondence

between the probable muscular affection as worked out *à priori*, and the actual affection as observed, is very remarkable, and goes far to show that the mode of advance of the disease is that which I sketched out in the earlier part of my paper.

II. In order to obtain additional evidence upon the point, I must, at the risk of appearing tedious, apply the same analysis to the nerves and muscles of the leg.

The nerves which we shall have to consider are :

- (1.) The anterior crural.
- (2.) The trunk of the great sciatic.
- (3.) The internal popliteal.
- (4.) The external popliteal.

(1.) *The anterior Crural.*—The *motor* branches supply the powerful muscles on the front of the thigh, and are very large.

The *sensory* branches are :

- (a) The middle cutaneous, which comes off high up in the thigh, and supplies the skin on the front of the thigh. The area supplied by it would therefore be attacked late, and disease would have a considerable distance to travel to reach a point where it might affect motor branches.
- (b) The internal cutaneous coming off high up in the thigh, and supplying the skin of the inner side of the lower third of the thigh and the inner side of the leg. The area supplied by it would be affected earlier than in the case of the last nerve, but disease would have a very long distance to travel before reaching motor branches.
- (c) The internal saphenous nerve also comes off high up in the thigh, and supplies the skin of the inner side of the lower half of the leg, and some parts of the foot. The area supplied by it would be early affected, but the distance to be travelled by disease would be very great before motor nerves would be affected.

From this we should expect that the motor part of the anterior crural nerve would long remain untouched, the distances to be travelled by the disease being very great

except in the case of the middle cutaneous nerve. This nerve, however, being affected late, and being still a considerable distance from its point of origin, disease would be little likely to reach the trunk of the anterior crural through it.

(2.) *The trunk of the great Sciatic.*—The motor branches supply the biceps semitendinosus semimembranosus, and partly the adductor magnus. The branches are given off, for the most part, in the upper half of the thigh. There are no sensory branches given off by the trunk of the nerve. We should therefore expect that, in consequence of the great distance to be travelled by the disease before reaching the above motor branches, the muscles supplied by them would remain intact.

(3.) *The internal Popliteal nerve and its prolongations.*—The motor branches of this nerve are :

- (a) Branches given off high up in the neighbourhood of the knee to the gastrocnemius soleus popliteus and plantaris.
- (b) Branches given off to the upper third of the leg to the tibialis posticus, flexor longus digitorum and flexor longus pollicis.
- (c) Branches in the foot to some of the small muscles of the foot.

The *sensory* branches are :

- (a) The external saphenous nerve arising a little below the knee, and passing down to supply the skin of the lower half of the back and outer side of the leg. The area of skin supplied by it would therefore be affected comparatively early in the disease, but the disease would have a long course to traverse before meeting with motor branches. The nerve also is very small, in comparison with the main trunk from which it comes.
- (b) The plantar cutaneous and other cutaneous branches to the foot coming off low down in the course of the nerve. The areas of skin supplied by them would therefore be some of the first to be affected, and the disease would have but a short distance to travel before reaching the motor nerves to the small muscles of the foot.

Thus then we should expect to find early and complete atrophy of the small muscles of the foot, but probably none of the large muscles of the back of the leg, belonging to either of the first two groups mentioned.

(4.) *The external Popliteal and its prolongation.*—The motor branches given off by the nerve are :

- (a) Nerves to the tibialis anticus, extensor longus digitorum and extensor proprius pollicis, given off by the anterior tibial in passing down the front of the leg.
- (b) Branches to the peronei muscles, given off by the musculo-cutaneous in its passage down the leg.
- (c) A branch in the foot to the extensor brevis digitorum.

The *sensory* nerves are :

- (a) Branches from the trunk of the nerve to the skin of the upper half of the back and outer part of the leg. This area would be affected at about the middle period of the disease, and the disease would have to traverse only a moderate distance before reaching the trunk of the nerve.
- (b) The main trunk of the musculo-cutaneous, which supplies the lower third of the front of the leg and the major part of the dorsum of the foot and of the toes. The area supplied by it would therefore be affected at the early part of the disease, but a considerable distance would have to be travelled before the muscular branches were affected—a distance considerably shorter in the case of the peronei muscles than of the other muscles of the front of the leg. The relative size of the sensory branches as compared with the motor approaches equality.
- (c) A small branch of the anterior tibial to the skin of the great and second toes, which would be affected very early in the disease, and up which disease would have only a short distance to travel before reaching the nerve to the extensor brevis digitorum, but a long distance before reaching the nerves to the larger muscles on the front of the leg.

Considering the external popliteal nerve as a whole, then, we should expect to find the extensor brevis digitorum to be atrophied early in the disease. We should also expect to find the peronei muscles atrophied earlier than the other muscles on the front of the leg, and much earlier than those at the back of the leg. As regards the relative periods at which the muscles supplied by the anterior tibial nerve would be affected, as compared with those supplied by the posterior tibial, the following remarks may be made: In the first place, according to Quain, the muscular branches given off by the posterior tibial nerve leave that nerve at a point higher up the leg than those given off by the anterior tibial. Speaking of the former, he says: "The muscular branches emanate from the *upper part of the nerve*, either separately or by a common trunk." Speaking of the latter, he says: "In its course along the leg, the anterior tibial nerve gives slender filaments to the muscles between which it is placed." According to Ellis, the branches to the deep muscles of the back of the leg may even come off from the internal popliteal nerve before it has become the posterior tibial. Thus in, at any rate, a considerable proportion of cases, the distance to be travelled by disease up the posterior tibial nerve will be considerably greater than up the anterior tibial, before motor branches to muscles of the leg will be met with.

Again, the anterior tibial nerve runs in the concavity of the angle formed by the leg and foot, whilst the posterior tibial runs in the convexity. When the general arrangements of the ankle are considered, I think it will be at once evident that the anterior tibial is shorter than the posterior tibial, which will still further increase the chances in favour of priority of attack of the motor branches of the anterior tibial.

We are thus brought to the conclusion, that the group of muscles supplied by the anterior tibial nerve will almost certainly succumb at an earlier period than the group supplied by the posterior tibial; whilst the group supplied by the internal popliteal is very unlikely to become involved, unless at a very late stage of the disease.

Treating the leg as a whole, then, we should expect all the small muscles of the foot to be atrophied. We should expect

the peronei muscles to be next affected ; and subsequently the other muscles on the front of the leg. Later still, the deep muscles at the back of the leg, and at a somewhat later period those of the calf. We should not expect to find any of the thigh muscles, either front or back, to be affected.

I regret that my notes are not sufficiently detailed to enable me to speak of the condition of all the muscles individually in the legs of my patient, but I have noted that all the small muscles of both feet were atrophied ; all the muscles on the front of both legs were completely atrophied ; the muscles at the backs of the legs were relatively healthy ; and the thigh muscles were apparently in their normal state of nutrition. I think, therefore, that I am justified in saying that the condition of the muscles of the legs bears out the conclusion arrived at by the examination of those of the arms.

I now pass on to a point of great interest, viz. the condition of the face. Here we saw that there was complete anæsthesia of the forehead and eyelids, the rest of the face and scalp being unaffected as regards sensation. Corresponding to this anæsthesia there was atrophy of all the muscles beneath the affected skin ; viz. the occipito-frontalis, the corrugatores supercilii, and the orbiculares palpebrarum ; all the other facial muscles being healthy.

Now here it is quite evident that the explanation offered above of the muscular affection in the limbs will not apply, for the sensory and motor nerve-supplies in the face have nothing in common, and hence no motor affections could be produced by disease travelling up the sensory nerves. On the other hand, the peculiar distribution of the atrophy points to an intimate relationship between the sensory and motor affections ; and points, moreover, to the conclusion that the atrophy was not due to disease in the trunk of the facial nerves, or even of the trunk of its temporo-facial division, a statement which a reference to the plate of the distribution of the nerve in Quain's 'Anatomy' will at once make evident.

In the face, however, we have to do with a muscular arrangement scarcely met with anywhere else in the body, viz. the muscles lie immediately beneath the skin, without the intervention of fascia or other thick tissue ; in fact, they may be

said to be practically united to the skin. Hence both they and the nerves connected with them would be peculiarly liable to become affected by any condition leading to infiltration or destruction of the skin. The muscular atrophy in this case is explained, then, by the direct extension of the disease to the muscles by contiguity, or possibly it may have resulted through the intermediary of their motor nerves, the peripheral termination of which may have been involved in the disease, in common with those of the sensory nerves. Possibly both processes have been at work simultaneously; but, however that may be, I think it may be considered as certain that this is the true explanation of the atrophy of these muscles. It is a point of considerable interest, that in this patient we have muscular atrophy produced in two widely different ways; the one being a direct affection from the skin by contiguity, and the other due to involvement of the trunks of the nerves supplying the muscles, by extension of disease up the sensory nerves from their peripheral terminations to their points of junction with motor nerves.

In conclusion, then, if, as I believe, this case may be taken as the type of an ordinary case of non-tuberculated anæsthetic leprosy, we may from it learn the method by which the nervous system becomes affected in this disease.

Leprosy is defined by Virchow to be a disease in which there is a production of a "new granulation tissue" that is effused into the fibro-cellular structures of the body. With the exception of the heart, pancreas, brain, and spinal cord, this neoplasm may invade the fibro-cellular coats and structures of all the organs. Clinical experience, however, shows that there are in the body two tissues which constitute the principal seats of election of the disease. These are the skin and the nerves. In many cases the skin alone is affected, the implication of the nerves being so slight that it may be left out of account. In some cases the nerves are implicated principally, the skin condition being of subordinate importance. In a large number of cases, however, both tissues are affected, the affection of the two tissues being relatively uniform over the body; or the skin affections may be most prominently marked in one part of the body, and the nerve affection in another.

There is nothing strange or unique in this varying elective affinity of the new growth. A similar variation is constantly seen in connection with new growths. Consider the case of syphilitic new growths, which will perhaps be found to be more nearly allied to the leprous neoplasm than any other form of morbid growth. There is scarcely any tissue in the body which may not be attacked by the syphilitic neoplasm; and yet how protean are the ways in which in given cases syphilis attacks the tissues, seizing greedily hold of some, and leaving others in their immediate neighbourhood untouched!

That the skin disease and the nerve disease are due to primary independent elective tendencies on the part of the original disease, and are not secondary the one to the other, whether the skin condition to the nerve disease, or the nerve condition to the skin disease (both of which statements have been made), can I think be proved by a reference to cases. A typical case of tuberculated leprosy is non-anæsthetic. Dr. Hillis lays stress upon this absence of anæsthesia in his general description of this variety of the disease. He says: "the palm of the hand also becomes fissured, horny and scaly from disease or malnutrition, *but not anæsthetic*" (the italics are his). Further on he says: "Anæsthesia ensues when the pressure of the blastema is more than ordinary," proving that it is only in exceptional cases of great thickening of the skin that it is met with.

Thus we see that the nerves are generally unaffected in those cases where the skin condition is most marked. On the other hand, in the typical anæsthetic form, though the skin is rarely quite free from deposit, yet it is present in comparatively small amount. Neither of these propositions could be true if there was any causal connection between the tuberculated skin condition and the nerve condition giving rise to the anæsthesia. But the independence of the two conditions is best seen in mixed cases of nerve and skin disease, as in the patient whose case I have narrated. In him the skin condition began on the trunk, and the patches of affected skin did not become anæsthetic; whilst on the other hand the nerve condition began at the extremities of the limbs, and, if we trust his history, independently of any skin disease. I think

therefore we may take it as proved, that the skin and the nerves constitute two independent seats of election for the original deposit.

There is, however, one way in which the skin will be liable to be affected by the nerve disease. It is well known how large an influence the nervous system has on the nutrition of tissues of all sorts ; and in proof of its influence on the skin, we have only to recall the conditions induced when a nerve has been permanently severed. The skin becomes degraded in every way ; it atrophies, its glands cease to secrete, and great care has to be exercised to prevent it from breaking down into low forms of ulceration. The same peripheral nerve-lesion, therefore, which causes the anæsthesia of leprosy, will cause atrophy of the skin, such as we saw had taken place in Mr. D. But if this be so with the healthy tissues of the skin, so will it almost certainly be the same with morbid growths, especially if of sluggish nature, like that of leprosy. Hence, supposing that a patch of skin were invaded by the morbid growth at the same time as the nerve supplying the patch was also attacked, the death of the nerve would lead to cessation of the skin growth. From this it will result, that the difference between tuberculated and anæsthetic leprosy will become accentuated ; for, in the former case, there is nothing to prevent the extensive deposit of the leprous neoplasm in the skin, whereas in the latter, even though there may be just as strong a tendency to the invasion of the skin, the simultaneous invasion of the nerves will hinder, and indeed almost prohibit, the skin affection in areas controlled by those nerves.

I will close my paper by suggesting a few points for investigation in future cases.

(1.) In mixed cases of tuberculated and anæsthetic leprosy, to notice accurately the difference between the eruption of parts of the skin where sensation is healthy, and that of patches of anæsthetic skin. With a view to proving the secondary influence of nerve disease in checking the morbid growth in the skin.

(2.) Is anæsthesia always preceded by modification in skin nutrition ? With a view to proving the independence of the nerve disease.

(3.) Does the primary invasion of anæsthesia ever give rise to patches of numbness corresponding to the supply of any named nerve? With a view to clearing up the question whether nerve-trunks are ever subjected to direct attack.

(4.) Does muscular atrophy ever take place without being preceded by anæsthesia? With a view to proving whether ultimate motor branches may be primarily affected, in a way similar to ultimate sensory branches.

(5.) Where muscular atrophy has taken place, to make a detailed examination of the various group of muscles. With a view to confirming or confuting the conclusions arrived at above, in reference to the mode in which their atrophy is produced.

(6.) An investigation of the deep sensibility of parts beneath anæsthetic skin, and a comparison of the deep sensibility of healthy muscles as compared with that of atrophied muscles.

GENERAL PARALYSIS OF THE INSANE. A STUDY OF THE DEEP REFLEXES, AND PATHOLOGICAL CONDITION OF THE SPINAL CORD.

BY WILLIAM CRUMP BEATLEY, M.D.,

Late Senior Assistant Medical Officer, Somerset County Asylum,

Wells.

THE study of the reflexes as a clinical symptom in nervous diseases, has of late commanded great attention. In 1875, Westphal and Erb each devoted a treatise to the study of this symptom, and the at present known ankle-clonus was then first described as the foot-phenomenon. Previous to this, Charcot, in his lectures at La Salpêtrière, pointed out in cases of paresis, or paralysis, of the lower extremities a certain trembling or trepidation, produced in the foot when it was caught by the point and suddenly turned back; since the treatise of Westphal, the foot-phenomenon has been carefully studied by various writers.

The study of the deep reflexes in General Paralysis of the Insane is exciting great interest among a small body of workers, their presence or absence tending to show that there is a close connection between general paralysis and other diseases, such as tabes dorsalis, and affording to some extent a basis for classification of a disease, which at present includes in a common description forms differing clinically and pathologically. Of the deep or so-called tendon-reflexes, the knee-phenomenon or patellar-tendon reflex, and the foot-phenomenon, or ankle-clonus, have been the most carefully studied. The purely reflex character of these phenomena is, however, at present strongly contested; from experiments and physiological data Westphal, Waller,¹ and others, conclude that the phenomenon is a local one. Be these opinions correct or not, the interesting fact remains,—that whatever increases or diminishes reflex action generally, increases or diminishes

¹ See 'BRAIN,' 1880.

the phenomenon under consideration. So far as our present knowledge extends, the following are among the most important causes which produce exaggerated and diminished reflexes.

(a.) Exaggerated reflexes.

1. Increased irritability of any factor of the reflex loop.
2. By far the most important condition, where cerebral influence is withdrawn from the spinal cord, by disease of some portion of the pyramidal tract, whether at the origin of those fibres in the cortex of the brain, in their passage through the corona radiata, internal capsule, pons, medulla oblongata, or in the spinal cord itself.

(b.) Diminished or abolished reflexes.

1. In *tabes dorsalis*, where the lesion is situated in the reflex loop, involving the fibres of the internal bundle of the posterior roots, or what Charcot calls "the inner radicular fasciculus."
2. In disease of the anterior grey horns, as in infantile paralysis, and in the advanced stage of pseudo-hypertrophic paralysis.
3. In disease of the posterior nerve roots.

By far the most important of these causes of changed reflexes are those :—1st, where there is lateral sclerosis of the spinal cord; this condition, it has been shown, by cutting off the inhibitory influence of the brain, gives rise to an increased reflex. 2nd, where there is posterior sclerosis of the spinal cord; in this case the reflex loop is directly interfered with. It thus follows that in considering the reflexes in any disease, one must at the same time take into account the pathology of the spinal cord. My own observations have been made with the following ends in view :—

- 1st. To determine the state of the reflexes in general paralysis.
- 2nd. To enquire into the connection between the reflexes and other clinical symptoms; and
- 3rd. To compare the state of the spinal cord after death with the state of the reflexes during life.

The following is an analysis of the condition of the deep reflexes in 65 cases of general paralysis which have come

under my notice; the observations were made at different times, and during different stages of disease, the results varying only in a way I shall subsequently describe. In some of the cases I am able to describe the pathological lesions found after death.

In the 65 cases examined, the knee-jerk was considered to be normal in 11, slightly exaggerated in 8, decidedly exaggerated in 18; it was diminished in 5, and absent in 18; in 5 cases the amount differed on the two sides. Ankle-clonus was absent in 47 of the cases, in one case it was not noted, and in 17 cases was present; being marked in 7, slight in 7, and in 3 cases present on one side and absent on the other. Of the 18 cases in which the knee-reflex is noted as decidedly exaggerated, 16 had ankle-clonus; the remaining case, in which ankle-clonus was present, had a slightly exaggerated knee-reflex. It thus appears that more than half of the cases examined showed marked alteration in the character of the tendon reflexes.

I will first consider those cases in which the knee-reflex, and with it ankle-clonus, was absent, omitting the cases where the knee-reflex was diminished, as I consider the question of diminution and slight exaggeration a personal equation. I am inclined to think that when the deep reflexes are absent, the mental symptoms are not so characteristic of general paralysis as in the opposite condition,—that of exaggerated reflexes; there may be delusions of an exalted character, but they are not so extravagant or obtrusive, and are, I think, apt to disappear early, leaving a more or less demented condition: in some this dementia is of the ordinary consecutive character; in others it is accompanied by a quiet expression of well-being and happiness, appearing in such expressions as, "I'm very well," "very happy," "as well as ever I was in my life," &c., generally attending this negative state, in an absence of that physical restlessness, which is, in my experience, a feature in those with increased reflexes. The patient is dull, sometimes sleepy, and will sit in one place for hours, staring vacantly in front of him. In common with other cases, they are subject to such accidents as apoplectic-form and epileptiform attacks.

The state of the pupil demands closer attention; in the majority of cases they were unequal, and in many irregular; in a large majority of the cases accommodation appeared perfect, whilst the reflex to light was absent or sluggish; there was, in fact, the Argyll Robertson pupil; the pupils were usually contracted, but in some cases normal in size, or even dilated.

I find no uniformity in the condition of the superficial reflexes, they may be normal or diminished.

The absence of a knee-jerk suggests a connection between the cases of general paralysis, now under consideration, and *tabes dorsalis*, and I have looked for other symptoms of the latter disease; in some of the cases, inco-ordination of movements has been detected, it has not, however, been a prominent symptom, being, as I believe, masked by the state of general paresis. Cases have been described in which ordinary insanity has been associated with *tabes dorsalis*; the insanity, however, following on the ataxy. A case in point is one described by Dr. Newcombe.¹ In this case, the ataxic preceded the mental symptoms by more than two years; in my cases, ataxic symptoms were only apparent late in the disease, and were never prominent. Cases, however, have been described in which the ataxia preceded the general paralysis.

The frequent presence of spinal myosis in general paralysis is an interesting symptom, common to it and to *tabes dorsalis*, and optic disc atrophy has been frequently observed in both diseases; it was present in some of my cases of the former disease. I have never observed skin eruptions, gastric crises, cephalalgia, diplopia, or marked anæsthesia of cerebral nerves. I have also enquired for the lightning pains of *tabes dorsalis*; and I may note that a case has recently been published by my late colleague, Dr. T. S. Sheldon,² in which he says, "the patient suffered from peculiar pains about the waist, and in the legs, which he described by darting his fingers rapidly forwards." Notwithstanding these negative observations, the absent knee-jerk points to an organic connection between the two diseases, and this indication has been confirmed in three cases in which

¹ 'BRAIN,' Vol. II. p. 134.

² 'Bristol Medico-Chirurgical Journal,' December, 1884.

I made post-mortem examinations, finding sclerosis of the posterior columns of the spinal cord, diagnosed during life by the absence of the knee-jerk.

CASE I.—John P., aged 55 years, a labourer, formerly a soldier, was admitted in January, 1883. Previous history negative, drank hard occasionally. On admission, patient is a small thin man, is somewhat excited. He has delusions of persecution and ill-treatment in the workhouse, saying the attendants kicked him to pieces, smashed his windpipe, so that he has not been able to speak properly since; also says he was scalped while in the Indian Mutiny. The pupils are irregular and unequal, the right being the larger; tongue protrudes slightly to the left, but is fairly steady. Delusion of his own greatness while in the army continued, and a general paresis of movements became evident.

In December he had an apoplecticiform attack, the legs being paralysed, the right appearing the worse. Three days afterwards, the general paresis was found to have increased, the right arm being weaker than the left. The pupils contracted and unequal; the deep reflexes were completely absent.

In January, 1884, the pupils are found to react but slightly to light, and actively to accommodation; he has great difficulty in co-ordinating his movements, and cannot stand with his eyes shut; he resists ophthalmoscopic examination. He has delusions of putrefaction; his mental condition is now one of dementia.

March.—He has had two attacks similar to above, becoming more helpless and paralysed; after this he deteriorated rapidly, becoming bedridden, and died on March 12.

Autopsy (twelve hours after death).—Body fairly nourished. Brain $43\frac{1}{2}$ oz.; the cranial cavity contained a quantity of fluid, vessels uniformly thickened, but free from atheroma. External convex surface of hemispheres affected to an extreme degree; wasting of the left hemisphere very marked; the meninges covering the frontal lobes and motor area are of a milky opacity.

Right hemisphere.—Meninges strip without adhesion from whole of convex surface, the gyri are atrophied, and diagrammatic in arrangement; they are much plumper than on the left side.

Left hemisphere.—Opacity and thickening of meninges marked to an extreme degree. Over fore-part of brain there are numerous small depressions filled with subarachnoid fluid; no adhesions over surface. Island of Reil much smaller on left than on right side. Pons wasted, crura small.

Spinal Cord.—To the naked eye the cord is tough and wasted;

there is distinct sclerosis of both posterior columns, all through the cord, and extending into the medulla oblongata. The pia mater over posterior columns is distinctly thickened. On section, the posterior columns are tough and gristly. Examined microscopically, the sclerosed tracts occupy the following positions. In the cervical region it is limited chiefly to the inner radicular zone. In the dorsal region it is a narrow line running from the centre of inner radicular zone, through the columns of Goll; and in the lumbar region, where the sclerosis is most extensive, it involves chiefly the inner radicular zone.

CASE II.—Joseph P., aged 32, a shoemaker. Admitted Nov. 6th, 1882. On admission, his condition was one of dementia, with excitement. He is said to have had delusions of wealth for some time, and to have been wet and dirty in habits. He is very demented, and unable to answer simple questions. The tongue is protruded in a very tremulous manner. The pupils are unequal in size, the right is ovoid in shape; his aspect is sullen. Locomotion is good. He is dirty in habits, drinking his own urine.

In *December*.—His speech is embarrassed, thick, blurred, and occasionally stuttering; he answers questions simply but coherently; he walks fairly well, but general tremor is very evident; he cannot write a connected letter. The deep reflexes are entirely absent.

January, 1883.—His dementia and general paralysis have very much increased, his articulation is worse. Is in bed with suppurating sores at all points of contact with bed-clothes.

His condition got rapidly worse, and he died on the 4th of February.

Autopsy (nineteen hours after death).—Body fairly nourished, covered with bedsores. Calvarium thick, dense and white.

Brain 43 oz. on first removal; after removing membranes and draining fluid off, it weighed $36\frac{1}{2}$ oz. There is much thickening and opacity of pia-arachnoid over frontal and parietal lobes, occipital portion fairly healthy; the arachnoid contains a quantity of brownish fluid.

Right hemisphere.—All the convolutions are extremely wasted, but especially the ascending parietal, and parietal lobules.

Left hemisphere.—Also wasted; the 1st, 2nd, and 3rd frontals most affected. The right temporo-sphenoidal lobe is soft and wasted. The membranes are adherent in one or two places to the parietal lobules on the right, and to the second convolution of the right temporo-sphenoidal lobe.

On section, cortex in frontal region very thin, lenticular

nuclei extremely small, ventricles much dilated, other parts wasted, brain oedematous. The floor of 4th ventricle was finely granular.

Spinal cord.—The cord is small, of fair consistence; no naked-eye changes were noted through whole of white columns of cord, membranes were not thickened. On microscopical examination, there is seen to be a marked sclerosis of the posterior columns, and other changes.

In lumbar region, the sclerosis involves the inner radicular fibres, extending thence downwards and inwards, occupying about the centre of Goll's column; it does not extend to the surface of cord, or up to the commissure; and a narrow tract bordering the posterior fissure is also unaffected; the lesion is most extensive in this region.

In the dorsal region, the sclerosis extends from the middle of the inner margin of the posterior horn, for a short distance downwards, occupying mainly the outer portion of Burdach's column, Goll's column being almost free. Under a high power the sclerosed tract shows nerve fibres varying in diameter, widely separated by a network of connective tissue. There is thickening of the vessels in the cord, and the following is the condition of a vessel running down the anterior fissure in the upper lumbar region: the wall is much thickened; there is a hyaline fibroid appearance, and the calibre is small. The inner layer of the pia mater is occupied by an almost homogeneous transparent material, which at certain points pushes itself amongst the nerve-fibres, towards which it presents a scalloped appearance.

In sections from this region, the pia mater bordering a portion of the lateral columns of the cord is much thickened, and translucent in appearance, it has a faintly granular look with numerous highly and refracting circular bodies scattered about in it.

CASE III.—This case presented the symptoms which I have noted as characteristic of absent reflexes, and the spinal cord, examined microscopically, presented the following lesion. There is a marked sclerosis of posterior columns through whole of cord, being chiefly limited to the inner radicular fibres; but in the lumbar region, nearly the whole of posterior columns appear to be involved.

Three cases are of course insufficient to prove a rule, still it is an interesting fact that, the knee-jerk being absent during life, sclerosis of the posterior columns and other

changes noted in the spinal cord, should have been discovered after death, and I am inclined to believe that this lesion will be found in most, if not in all, cases of general paralysis, in which the knee-jerk is markedly absent. We must remember that there are cases of one-sided-brain disease, in which the knee-jerk is absent, and in which there is no lesion of the cord. Such a case is published by Dr. Mackenzie in 'BRAIN,' Vol. VI. p. 222, but such lesions as would lead to this result, may be excluded from general paralysis, the cortex lesion in which disease would rather suggest a descending degeneration. Why posterior sclerosis, an ascending lesion as we are taught, should be associated with general paralysis, is a question I am not prepared to answer; that it is not a mere coincidence may be inferred from the large proportion of cases in which the knee-reflex was found absent, and from the direct evidence of other histologists.

One of the most interesting observations on this subject is by Dr. Dowse:¹ he draws attention to some of the disturbances of reflex action common to general paralysis of the insane, and to tabes dorsalis. He says, "the patellar tendon-reflex may be absent in both diseases," and he is inclined to believe when such is the case in general paralysis, some change in the posterior columns of the cord has occurred. He gives Westphal's view of pathology, namely, that no direct relation exists between the morbid process in the cord and the morbid process in the brain, but that they are the expression of an excessive proclivity to disease of the nervous system. Westphal describes a sclerosis of the posterior columns similar to that in the cases I have related, and designates such as the "tabetic form" of general paralysis, a tabic gait being present. In my experience this form of gait is present in some cases. It was marked in the case of John P., Case I., but was not at all marked in the other cases, and its absence has been noted by other observers.

Exaggeration of the patellar tendon-reflex, associated in a large number of the cases with ankle-clonus, was found in twenty-six of the sixty-five cases examined. The ankle-clonus was marked in some of the cases, and slight in others; and in

¹ 'Brit. Med. Journ.' vol. i. 1882.

the cases in which it was present, varied considerably in amount at different periods, being at one time easily obtained, and at others with difficulty, or not at all. In my experience, the cases which present these exaggerated reflexes are the most typical examples of the disease, corresponding much more nearly with the classical description than do those in which the reflexes are absent. The delusions of grandeur are more constant and obtrusive; they possess great wealth, can perform great deeds, devise plans of an impossible and gigantic character, and invest mean surroundings with charms that do not exist. They are usually loquacious, and restless in their movements; there is a notable impairment of the finer muscular actions—those of the mouth, lips, and fingers—and in speech they slur their words. In the earliest stages there is a peculiarity in the gait of marked cases, the step being elastic and springy, and they are constantly on the move. In shaking hands, the act is exaggerated, the grip being convulsively firm. Savage has noted a tendency to fix the eyebrows, before they begin to speak, and in one case under my care this was well marked. These cases show tendency to fatformation; the face becomes bloated and expressionless; ultimately there may be a rapid loss of flesh, and the paralysis becomes more marked; generally there is a difference in the amount of paralysis of the two sides. The condition of the pupils is not constant; in many there is irregularity of outline, and in most cases inequality; and in a few cases loss of reaction to light, whilst accommodation remains. Contrary to expectation, Savage has found in these cases optic-disc atrophy. The presence of an exaggerated deep reflex is not so valuable a sign of spinal-cord lesion as is the absence. Some observers state that both increased patellar reflex and ankle-clonus can be present without such lesion, as is the case sometimes in hysterical paraplegia.

The patellar reflex, being a phenomenon usually visible in health, and varying in amount in different individuals, there is always some difficulty in determining an abnormal amount. When ankle-clonus, however, accompanies increased patellar reflex, it is no doubt a grave reason for suspecting sclerosis of the pyramidal tract, more especially in such a disease as

general paralysis, in which disease of the cortex of the motor convolutions is often a marked feature. In the case of general paralysis, an interesting question arises as to the pathology of sclerosis of the pyramidal tract; as a rule, interruption of the tract at some given point, by injury or disease, starts the descending degeneration. Where does this interruption take place in general paralysis? In the case of G. J. F. (Case IV.) there was a double lateral sclerosis; but the most careful search failed to make out any break of continuity in the pyramidal tract. The meninges were not adherent to the cortex, but there was marked atrophy of the convolutions in Ferrier's motor area, from a portion of which, we are taught, the tract originates.

It seems to me that there are but two ways of explaining the presence of lateral sclerosis in such cases.

1st. That the atrophy of the pyramidal cells, probably the larger ones in the third layer of the cerebral cortex, is sufficient to give rise to a descending degeneration of the pyramidal fibres connected with them; in this connection I quote from Charcot, 'On Localization of Cerebral and Spinal Disease:' "It is not yet proved that destructive lesions, limited to the grey matter of the Rolandic region, are productive of secondary degenerations, nevertheless some facts taken from the pathological anatomy of general paralysis tend to show that this is really the case."

2nd. That, as explained when treating of posterior sclerosis, the sclerosis of the lateral columns may be part and parcel of a general increase of fibrous tissue in the cerebro-spinal axis.

I see no difficulty in the latter view, if we may believe in the existence of a primary lateral sclerosis. Westphal believes that in general paralysis only has primary lateral sclerosis yet been described; but Flechsig interprets the sclerosis as consecutive to cortical lesions. I am inclined to accept Flechsig's views, as lateral sclerosis occurs too frequently in general paralysis to be regarded as a mere coincidence.

CASE IV.—G. J. F. Admitted September 1882. In January, 1881, he had a paralytic attack, from which he has never completely recovered; since then his mind has gradually become affected. His wife says he was walking home during a snowstorm when first attack occurred. After two months he had a second attack, on recommencing work, and has had two or three attacks since. With each attack he loses power of speech, and in left arm and leg. He has had delusions at times.

On admission.—He has a wild vacant expression, is quite incoherent, and cannot answer simple questions. Speech drawling and thick; tongue protrudes with a jerk; pupils unequal and irregular; there is much want of power in left arm and leg. Tendon reflexes much exaggerated, more particularly in left side. There is slight external strabismus of left eye.

October.—Is failing fast; general paresis increased; cannot stand; tendency to bed-sores.

Nov. 21.—Is in bed, both legs paralysed, but he moves them a little; uses arms apparently equally. As legs lie flat on the bed, a tap of right patellar tendon causes contraction of quadriceps muscle, similar to that produced by tapping the muscle itself. On left side a convulsive kick of limb follows. Sole reflexes seem normal and equal. Sensation is certainly deficient. Patient more demented.

Jan. 24, 1883.—Has gradually got more paralysed and weaker. This morning had right-sided convulsions; at noon there is no convulsion, but firm contraction in flexors of right arm; eyes turned to the left. Patient unconscious.

Jan. 28.—Died from pneumonia of left lung.

Autopsy.—Calvarium thickened. There is a bluish opacity and thickness of the basal vessels. Brain 50½ oz., very cedematous; pia-arachnoid thick and opaque over frontal lobes and motor area; the gyri are much wasted on both sides. No coarse lesion discovered on section.

Spinal cord.—To the naked eye there is marked sclerosis of both lateral columns, extending through whole of cord. Examined microscopically, the sclerosis occupies the usual position in the lateral columns, viz. what Charcot calls, the “crossed pyramidal fasciculi.”

CASE V.—J. V., aged 36, married. A builder's accountant.

Admitted Oct. 8th, 1883. Supposed causes, heredity and business failure. First symptoms, noticed six months ago, being loss of memory and mistakes in his work.

On admission.—Is a well-built, intelligent-looking man of medium height; has a self-satisfied expression. He talks coherently

and volubly ; his memory is good. He has the most extravagant delusions ; says he is the richest and most wonderful man living, that he has built palaces of diamonds, and invented a machine for paving the streets with gold at one penny a foot, &c. In conversation he occasionally stammers and slurs his words ; has great difficulty in pronouncing such words as "statistical." His tongue and lips are tremulous, and he protrudes his tongue with a sudden jerk. There is capillary congestion over both malar bones, and in speaking he frequently fixes his eyebrows. In walking, his gait is springy and firm, he takes long strides, and walks a good deal on the toes. In shaking hands, he grips the hand firmly and convulsively. There is evident paresis of the fine muscular movements ; his finger movements are awkward ; he is a fair musician, and when playing the piano, he leaves out notes and slurs them. His pupils are unequal, the left being the larger ; they react to both light and accommodation. His patellar tendon-reflexes are both markedly exaggerated, and there is ankle-clonus on both sides.

May 1884.—Has the same exalted delusions. The general paralysis has increased ; knee-reflexes still exaggerated, but ankle-clonus obtained only with difficulty. He is extremely restless ; constantly on the move ; in walking droops left shoulder, and drags left foot slightly.

July.—Bodily weight has increased. Still has exalted delusions, but is quieter ; does not talk so much ; is irritable, and easily displeased.

Sept.—Is getting feeble, losing flesh. Mental condition more one of dementia. Knee-reflexes still exaggerated ; ankle-clonus absent.

Here, unfortunately, I lost sight of the case ; but I am informed by my late superintendent, Dr. A. Law Wade, that he died in January 1884, after a large number of epileptiform convulsions, chiefly on right side. The brain showed the usual lesions of general paralysis, and the spinal cord showed a later posterior sclerosis in lower dorsal region, with an enormously dilated central canal.

CASES VI. and VII.—These cases showed symptoms which I have previously noted as characteristic of an exaggerated deep reflex. I will therefore only give the condition of spinal cord, found after death.

CASE VI.—G. C. To the naked eye, there is a marked sclerosis of both lateral columns in the usual positions ; the cord is firm and tough. Examined microscopically, the sclerosis is visible through

extent of cord, but chiefly in dorsal region. Here and there are separate plates of connective tissue, and in one series of sections in the dorsal region there is a patch in posterior column on right side.

CASE VII.—J. S. Cord firm, no wasting. To the naked eye, there is sclerosis of lateral columns in usual position; a fresh section shows same condition.

So far, therefore, as my observations have gone, it would appear that in the state of the deep reflexes we have valuable indications of the state of the cord in general paralysis of the insane. They also show that disease of the spinal cord exists far more frequently than has hitherto been understood; that in some cases the morbid process may commence in the brain, and in others terminate there.

A provisional classification can be made from the state of the deep reflexes as follows:—

1st. A tabic form, in which there is posterior sclerosis of spinal cord. 2nd. The paralytic form, in which there is lateral sclerosis of the spinal cord; and, 3rdly. A form which is cerebral rather than cerebro-spinal, physiological tracts in the cord not being affected.

Of the latter class, in which the reflexes are not altered, I have made post-mortems in eight cases. In none of these did I find a fasciculated sclerosis; the general symptoms follow more closely the classical description of the disease.

Clinical Cases.

CASE OF BRACHIAL MONOPLÉGIA, DUE TO LESION OF THE INTERNAL CAPSULE.

BY A. HUGHES BENNETT, M.D., AND C. M. CAMPBELL, M.D.

GENERAL P., æt. 80, served his full time in India, but had retired, and lived in Europe for upwards of twenty years.

According to the statements of his relatives, his temper and disposition had changed for the worse during the past few years, —but his general health had been good. He suffered, however, from marked muscular tremors of both superior extremities, for an uncertain period, before he came under observation.

On November 17th, 1884, he dressed himself, went through several corridors, and descended in the lift to the saloon of the hotel in which he was staying.

When first seen, he was seated at breakfast, making partially successful attempts to feed himself.

He complained of difficulty of speech; and had remarked on getting up in the morning that his mouth was drawn to the right side. There was marked paresis of the left face and tongue, but no other part of the body was affected. The patient was ordered to go to bed, and avoid stimulants and excitement of any sort.

When next seen at 6 P.M., it was stated that an hour previously he had got up, and fallen insensible on the floor. He was now partially conscious; but the paresis of the left face was more marked, his speech was quite unintelligible, and voluntary motion of the left upper extremity entirely gone.

For some days, deglutition was so imperfect that the patient was chiefly fed per rectum. Urine was occasionally passed involuntarily, and the power of the sphincter ani was diminished.

On December 1st, articulation and deglutition were much improved, and facial paresis less marked. The movements of the eyeballs, vision, and the special senses were normal. The power of walking was not tested, but when in bed, no loss of power in the lower extremities could be detected. The knee-jerk was slightly more marked on the left side.

Subsequent examination showed the face to be normal. Although locomotion was feeble, there was no evidence of special paresis of the left leg. The arm remained as before, completely paralysed,—the muscles flaccid and without trace of rigidity. The sensibility was everywhere normal.

For about four weeks the general condition improved. About this period intelligence became clouded; the patient talked Hindustanee, or Italian, without apparently being aware that those about him did not understand him; he became lethargic, and unable to fix his attention. This condition continued to intensify, until he died on January 20th, 1885, without developing any new special symptoms.

Post-mortem Examination.—Permission was obtained, on the 21st, to examine the brain only. The dura mater was found thickened throughout, and in many places strongly adherent to the skull; the arachnoid and pia mater were deeply congested, opaque, and thickened, and there was considerable sub-arachnoid effusion. All the arteries noted were thickened and atheromatous. The convolutions generally were flattened, and soft to touch, but otherwise normal.

A series of vertical sections was made across the brain, which showed its substance to be slightly congested, and softened, but otherwise normal, except at the spot about to be described.

A vertical section, in the direction of the fissure of Rolando, through the middle of the right ascending parietal convolution, disclosed a limited defined softening, at the upper part of the internal capsule, or foot of the corona radiata. The softened area was about the size and shape of a horse-bean, though flatter, and occupied the position indicated in the accompanying diagram (Fig. 1, p. 80). It measured $\frac{5}{8}$ of an inch in its long, and $\frac{3}{8}$ of an inch in its short axis, but not more than $\frac{1}{8}$ of an inch in thickness antero-posteriorly. Its upper edge just touched and slightly involved the outer and upper border of the caudate nucleus. From thence it stretched obliquely downwards, across the internal capsule, to the upper margin of the lenticular nucleus, the apex of which it very slightly invaded. The caudate and lenticular nuclei, and the neighbourhood, were otherwise normal.

COMMENTARY.—All the circumstances of the case during the life of the patient led to the assumption, that the lesion existed in the middle parietal fasciculus of the centrum ovale; in the triangle of white conducting substance, the base of which is the cortical substance of the ascending parietal convolution, and the apex, the upper part of the internal

capsule behind its knee. There was no evidence as to the exact level at which the interruption occurred.

After death, a limited, flat, and perfectly circumscribed softening was found occupying the upper part of the internal capsule, extending between the caudate and lenticular nuclei; its situation corresponding to a line of vertical section through the middle, and in the direction of the ascending parietal convolution. This had caused complete, universal, and permanent paralysis of the left superior extremity only; the antecedent and accompanying immobility of the left face being transitory, and weakness of the left leg, if it existed at

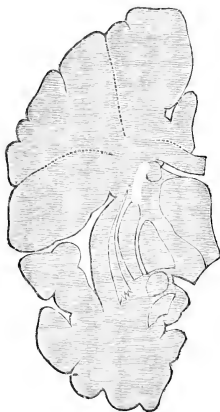


FIG. 1.—Vertical section of Hemisphere through the middle of the parietal convolution. The white patch marks area of softening between caudate and lenticular nuclei.

all, being equally so, as none was observed four weeks after the attack. The chief interest of the case lies in these phenomena, which differ from those of common general hemiplegia, due to more diffuse disease of this region. In all such cases, including the present one, the destruction of tissue is probably due either to hæmorrhage from, or to obstruction of a branch of the lenticulo-striate artery. But in the present instance the symptoms, at first somewhat diffused, doubtless from shock, subsequently remained strictly limited to the arm, the result of the flat patch of softening already described, stretching across the capsule in a nearly transverse direction.

The fact that such a lesion should cause permanent paralysis of the left superior extremity only, leaving the left face and leg unimpaired, shows that the fibres conducting motor impulses from the cerebral cortex run in separate ribbon-shaped bundles, through the internal capsule; and that their flattened surfaces are at right angles to an antero-posterior section of the cerebrum.

Assuming the same conditions for the other motor centres of the body, we may hazard a general idea of the arrangement of

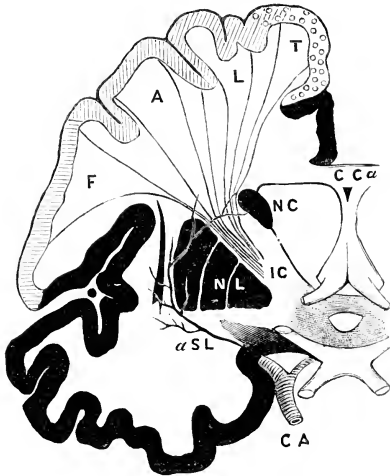


FIG. 2.—(After Horsley.) Vertical section of Hemisphere through the Fissure of Rolando, showing relative position of motor centres, F. Face, A. Arm, L. Leg, T. Trunk. The fibres run from motor centres into internal capsule between the nuclei of the corpus striatum and the "artery of hemorrhage" (Duret). C. C. Corpus Callosum. N. C. Caudate nucleus; N. L. Lenticular nucleus. C. A. Internal Carotid Artery; a. S. L. Lenticulo-Striate Artery. Cf. fig. page 132.

the conducting fibres, between the cortex of the brain, through the pyramidal tract to the internal capsule. Round the fissure of Rolando are situated those centres which preside over the voluntary movements of the trunk, leg, arm and face of the opposite side of the body. This sulcus lying in an oblique direction, downwards, forwards, and outwards, makes it evident that the various areas lie not only at different levels, but in different positions as regards their

antero-posterior and lateral situations. Thus the centre for the trunk is the most superior, posterior, and internal. Immediately contiguous is the centre for the leg; next in order, that for the arm; next and last, that for the face, which is the most inferior anterior and external. The four bundles of conducting fibres, descending from these centres in the above order and position, in a downward and obliquely inward course, may be compared, when viewed from the front, to four rays of a half-opened fan. These converging in this attitude toward the upper part of the internal capsule enter its knee, and as they do so, change their direction. Preserving their

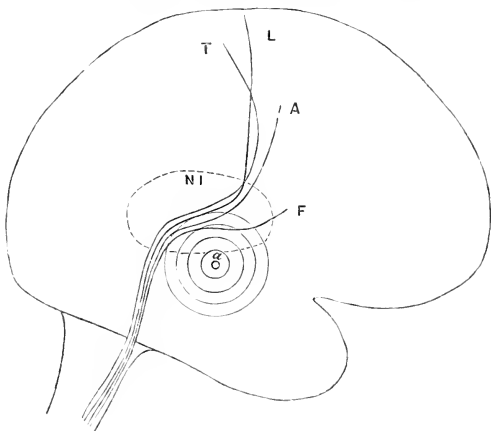


FIG. 3.—(After Horsley.) Motor centres as seen from the side. F. A. L. T, as in Fig. 2. N. I. Lenticular nucleus, indicated by the dotted line. The fibres of the direct pyramidal tract are seen passing down to the internal capsule, arranged first in a superior-inferior order, and afterwards in an antero-posterior order.

relative stations, they become twisted from their former oblique position, to one completely antero-posterior. So that what was external and in front, becomes anterior; and what was internal and behind, becomes posterior. The half-opened fan is now shut, and its rays, which before were seen obliquely in front, are now seen from the side. In this last position, all the series of fibres seem to pass through the internal capsule in well-defined ribbon-shaped bundles, those for the face being the most anterior, behind which in successive order we find those for the arm, leg and trunk

That some such arrangement existed, had already been suggested, especially in a recent paper by Mr. Victor Horsley on "Substitution"¹ The present case supports these hypothetical views, by showing that a limited, flat, transverse lesion of the internal capsule can cause paralysis of the superior extremity, without destroying the mobility of the face and inferior extremity. In short, the second anterior ray of the

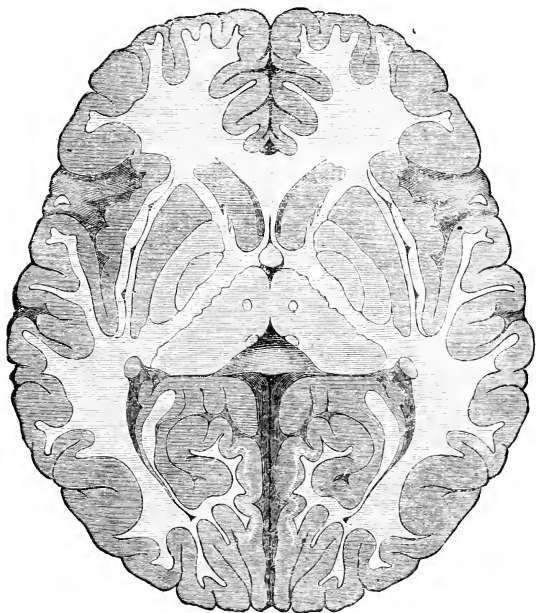


FIG. 4.—Horizontal section through Basal Ganglia, showing the entire horizontal length of the internal capsule. The white patch on the right side behind the knee represents the probable situation of the softening, the other aspect of which is seen in Fig. 1.

fan may be divided and destroyed, those in front of, and behind it, remaining intact.

It would have been a point of interest to have demonstrated the exact antero-posterior position in the internal capsule which this limited lesion occupied. The whole brain, however,

¹ 'Lancet,' July 5, 1884. See also Reviews and Abstracts in this No. of 'BRAIN.'

could not be obtained for further investigation, as permission for the autopsy was only granted on condition that the organs should be returned. A small section containing the lesion was however retained, which establishes and preserves the size and situation of the lesion, as demonstrated by a *vertical* section; but its exact position, on *transverse* section, could not be obtained.

The following experiment was therefore made on a normal brain. A vertical section having been made in the same position as that in the specimen, viz. down the centre of the ascending parietal convolution in the direction of the fissure of Rolando, and a transverse section through the middle of the internal capsule, through the basal ganglia, it was found that the spot at which they bisected each other, was a little behind its knee (Fig. 2).

This is where the lesion, presumably, would have been found.

AN UNUSUAL CASE OF ATHETOSIS.

BY SEYMOUR J. SHARKEY, M.B.,

Assistant Physician and Joint Lecturer on Pathology at St. Thomas's Hospital.

G., ET. 24, came under my care as an out-patient at St. Thomas's Hospital, on March 26th, 1884.

Until the patient was three years old he was quite healthy; he was then suddenly attacked with loss of power all down the left side, accompanied by repeated and prolonged convulsions.

As far as he knows, the movements of the limbs and face on the left side gradually followed the loss of power, and have gone on increasing ever since; at any rate, they have done so as long as he can remember. When between four and five years of age he was taken to Great Ormond Street Hospital, where his parents were told that the movements were so slight, that he would get over them.

About three years ago weakness and movements came on in the right leg, and he went to Guy's Hospital, where he was told that nothing could be done for him. He then consulted a homœopathic chemist, who gave him "strengthening medicine," and his right leg got strong again, and the movements ceased in three months.

A month ago the weakness and movements commenced again in the right leg, and they have gradually increased. The arm is said to be free from them.

The patient has never had any other severe illness, has not suffered from headaches, and has done his work well as a carpenter until the last six months. During this time the movements of the left hand have been so bad that he has been unable to hold the nails with accuracy, though he can hammer well enough with the right arm.

He says he has never had any defect of sight or hearing.

On rough examination, vision seems normal, and he hears the tick of a watch a long way off.

There are movements of the greater part of the muscles on the left side of the body, and they cease during sleep. There is no anaesthesia.

The tongue moves a little irregularly, as it does in chorea. Speech is drawling. The muscles both of the left and of the right side of face contract too strongly when the patient attempts to speak, so that a kind of grimace results, in which the lines at both angles of the mouth and the naso-labial furrows are very deeply marked.

These are spasmodic, more or less regular movements of the neck

to the left, and the shoulder is rhythmically raised and jerked slightly forwards and backwards.

One can also see the serrations of the left serratus magnus contracting rhythmically. The abdominal muscles are harder on the left side than on the right, but there are no evident movements in them.

There are slight spasmodic movements in the muscles of the left arm; but they are only slight. The hand, on the contrary, is markedly affected.

There is constant more or less rhythmical flexion and extension of the fingers, mainly at the metacarpo-phalangeal joints, with irregular flexion and extension of the phalangeal joints, and occasional separation of the fingers. In the left leg there are similar slight movements of the muscles, with marked movements of the toes, and most marked of the great toe. They are tremulous flexions and extensions of the toes; but the flexor longus hallucis is constantly contracted, so that the great toe is always riding over the others, and its proximal phalanx is usually at right angles to the metatarsal bone. In the right foot the movements are similar, but not so marked. In the right hand only the little finger moves, and that so slightly, that the patient had not noticed it. The grasp of the right hand, though stronger than that of the left, appears to be deficient in power; the patient says, however, that he has never noticed that it was weak. There is neither atrophy nor hypertrophy of the muscles of the limbs. The patellar reflex is unnaturally brisk on both sides, but there is no ankle-clonus.

The patient seems very intelligent, and says that his memory and mental power are very good.

Fundus of eyes and fields of vision natural.

Urine contains no albumen.

June 7th.—Admitted into St. Thomas's Hospital.

While an out-patient he had been treated with Easton's syrup, and subsequently with phosphorus in pills, but without any good result. Indeed the movements on the right side had rather increased.

On admission.—The right middle and ring, as well as the little finger, present rhythmical movements, and the wrist is turned slightly to the ulnar side. There are, in addition, occasional contractions of the right sterno-mastoid muscle.

The abdominal muscles are hard on both sides, though more so on the left than on the right, and the divisions of the recti can be clearly seen.

The patient sleeps badly, but he cannot say why.

While in hospital for several weeks he was treated with the continuous current and improved very much. The movements diminished decidedly on the right side, but never ceased altogether. The patient expressed himself as very much better and able to do many things which he formerly could not do. He was then sent away to the country.

Remarks.—Oulmont, in his Monograph on Athetosis, describes two forms of the affection, one unilateral, the other bilateral. The former, hemiathetosis, he says is an affection generally symptomatic of a cerebral lesion, and consists essentially of involuntary movements, habitually continuous, slow and exaggerated, and limited to the hand and foot of the paralysed side. Bilateral athetosis, on the other hand, he describes as an affection always primary, consisting essentially of involuntary, slow movements, involving the two hands, or the hands and feet together, and sometimes the two sides of the face. Several characters distinguish it from unilateral athetosis: first, its origin; for it is a primary affection of the earliest childhood, or perhaps congenital. Secondly, its localisation; for it involves the extremities of both sides of the body, and sometimes also both sides of the face. Finally, the movements are much more feeble than in hemiathetosis, often intermittent, and occurring only during attempts at voluntary movement. Moreover, it is usually associated with idiocy.

The case described in this paper must be placed in the same category as those described by Oulmont as hemiathetosis. For it was not congenital, but originated in a sudden attack of illness which resulted in hemiplegia: it remained for many years unilateral; and the intellectual capacities of the patient have never been in the least degree deficient.

The peculiarities of the case which seem to make it worthy of record are, first, the gradual supervention of movements in the muscles of the right side of the body after the disease had been confined for eighteen years to the left: and secondly, the great number of muscles which are affected in addition to those of the hand and foot.

Oulmont attributes the affection to disease of what he calls the "Athetotic fibres," which he supposes to exist in the posterior part of the internal capsule in front of and outside the sensory tract. Such a localisation of the disease appears to be very unlikely. It may, however, be presumed that in all probability the affection in the present case resulted from some coarse and sudden lesion, possibly hæmorrhage, in some portion of the right hemisphere situated more deeply than the cortex.

It would be exceedingly interesting to know by what means, or by what route, the movements spread to the opposite side, whether by means of the commissures of the brain, or by the direct pyramidal tract, or by fibres or cell-processes connecting the large motor cells of the anterior cornua of the spinal cord. But there do not appear to be sufficient data for determining this point; the case is therefore simply recorded, and speculation is left for the reader.

CASE OF PERFORATING TUBERCULOSIS OF SKULL WITH CEREBRAL SYMPTOMS.

BY WALTER EDMUNDS.

PATIENT was a boy, aged 14, of a rather delicate family, one of his sisters having died of hip disease.

Eighteen months before admission, he had an attack of peritonitis which was thought to be tubercular.

About the same time, there formed on the left side of the scalp over the parietal bone a cold abscess, accompanied by much headache; this abscess was twice aspirated, and thick pus drawn off each time.

Some six months later he complained, on and off of numbness in the right arm and hand, and four months later the abscess in the scalp began to fill again.

Two days before admission he had a fit—the first he had ever had. He felt giddy, fell, became unconscious, foamed at the mouth, bit his tongue, and passed water involuntarily; both legs were convulsed, and the right arm clenched and drawn up.

State on admission: A pale but well-nourished boy; on the left side of the scalp is a soft fluctuating tumour, from which a mixture of blood and pus can be drawn out with a fine syringe.

The grasp of the right hand is much weaker than that of the left; the right leg is also weaker than the left, but the difference is only small; there is, too, slight right facial paresis.

Sensation everywhere normal.

Ophthalmoscopically both discs are slightly swollen and their margins blurred; the veins kinked, and white lines along the vessels. No hæmorrhages.

Temperature normal. Sleeps well. No headache. No albumen or sugar in urine. He never vomited, and never noticed any defect of sight. Knee-reflexes normal.

Under ether the abscess was laid freely open. At its base was found a piece of diseased bone of oval shape, measuring seven-eighths by five-eighths of an inch; it was depressed on one side, and nearly completely separated from the surrounding bone, so that with an elevator it could be lifted up and removed.

It comprised the whole thickness of the skull, the dura mater being exposed, and the pulsations of the brain seen.

The wound was dressed with iodoform, and (except for the formation of a diminutive aneurism in the scalp which had to be opened) progressed uniformly well; there was, however, some suppuration with loss of substance on the surface of the brain.

He had two fits subsequently; one two weeks, the other eight weeks, after the operation; in both of them the head was turned to the right, he had general convulsions and foamed at the mouth.

The discharge from the openings in the skull gradually diminished, and the scalp covered over the edges of the skull,



(From a Photograph.) The lines indicate the great longitudinal fissure, the two branches of the fissure of Sylvius and the fissure of Rolando. The cross is at the parietal eminence.

so that when he was discharged there was an opening in the scalp and skull through which the brain could be seen. A silver plate was made to protect this.

The inflammation of the optic discs gradually subsided, but that of the right eye is not yet quite normal.

In about four months he was sufficiently well to get up and walk about; the chief thing remaining wrong with him, besides the opening in the skull, was the weakness of the arm and hand, the strength of the right hand being only a third of that of the left.

Shortly before he went out, I drew out on the boy's shaven scalp with the assistance of my friend Dr. Reid, the lines

he has given, for indicating as nearly as possible the positions of the cerebral fissures. (See woodcut.)

The opening in the skull was seen to be half-an-inch in front of the middle of the fissure of Rolando; but as the superficial ulceration of the brain extended for a short distance backwards from the opening, the middle of the ascending frontal and ascending parietal convolutions were affected. Probably the centre marked VIII. and one of the centres marked x. by Ferrier were involved; these are concerned, he says, with the flexion of the forearm and the movements of the hand and wrist.

This case is recorded partly as an example of a somewhat rare disease, "Perforating Tuberculosis of Skull," an affection which has been carefully described by Volkmann in the 'Centralblatt für Chirurgie,' 1880, No. I, and partly because it shows a coincidence between the localising cerebral symptoms and the measurements on the surface of the scalp.

CASE OF PACHYMENINGITIS CERVICALIS HYPERTROPHICA.

BY C. W. SUCKLING, M.D. (LOND.), M.R.C.P.,

Physician to the Queen's Hospital, Birmingham.

W. B., a boy aged 18, was admitted into the Workhouse Infirmary, May 25th, 1883, with paralysis and tucking of both legs.

When five years of age, he fell down some steps on to the back of his head. Ever since, he has had an internal strabismus of the left eye. Another fall two years previously to his admission, he says, caused the paralysis; in his opinion the paralysis commenced shortly after.

The patient had been in the General Hospital for twelve months before he was brought to the infirmary, and while there, both arms and legs were paretic and rigid. The paralysis began in the right arm; the onset was gradual, and attended with numbness, tingling, and loss of sensation.

There was great pain in the neck and scalp, for which a seton was introduced, and the neck was rigid, movement causing great pain. The pain was along the great occipital and third cervical nerves, upward towards the head, also down the shoulders.

Two or three months after the paralysis of the right arm, the left arm, both legs and back, began to get weaker, until the patient was completely paralysed.

The arms were rigid, with extreme flexion of the fingers and wrists. There was no paralysis of the bladder or rectum.

Five months before admission (in 1883) he began to recover the use of his arms, and they have been getting better ever since.

There was never any facial paralysis, or any cerebral affection or disease of the cranial nerves, no vertigo, delirium, or vomiting.

The legs were weak when the arms were affected. At first they were flaccid, but they gradually began to get stiff, and contracted and trembled. All four extremities were paralysed together, but the arms got better while the legs got stiff.

When admitted, the patient was much emaciated, had spasmodic internal strabismus of the left eye and torticollis, the

head being twisted to the right. He could rotate his head more to the right than to the left.

No tenderness could be elicited by percussion over the cervical spines, and there was no fever or any sign of scrofula.

Both legs were paralysed and flexed at the hips and knees, with adduction of the thighs.

The arms had recovered power, but were still weak. There were no sensory disturbances.

Ankle-clonus and front tap contraction were markedly present in both legs. On account of the contracture, the patellar reflexes could not be obtained.

The plantar reflexes were present; the cremasteric, abdominal and epigastric absent.

There was no sensory disturbance in the lower extremities, and no marked wasting.

The optic disc and fundus on each side was normal. The patient was treated with iodide of potassium, and counter-irritated over the cervical spine, and when examined in July 1884, was much better, could rotate the head more, could use the right leg well, and there was little rigidity on this side, but still ankle-clonus.

The left leg was still tucked, and wasted with increase of reflexes.

He was well fed, and spent as much time in the open air as possible, going about on crutches. A shot bag was suspended to the ankle of the left foot, to diminish the contracture. On the 14th of March last, the patient, wishing to leave, was again examined. The chin was still slightly turned to the right, the reflexes (bicipital, &c.) in the upper extremities well marked, but there was no rigidity, and the grasp on both sides was very powerful.

Ankle-clonus had disappeared, and the contracture of the left leg much diminished. He could not, however, put the left heel to the ground, there being talipes equinus. The left leg was considerably wasted. The circumference of the left calf was $10\frac{1}{2}$ inches, of the right, 13 inches.

This wasting can only be accounted for by supposing that an extension of the morbid process in the crossed pyramidal path to the anterior cornua has taken place.

The electrical reactions of the nerves and muscles of the left leg are normal.

As to the diagnosis of the case:—

Amyotrophic lateral sclerosis is excluded by the marked sensory disorders and progress of the case to recovery.

A transverse myelitis, by the great predominance of the irritative phenomena, and absence of anaesthesia, and complete paralysis of the leg preceding the rigidity. Progressive

muscular atrophy by the presence of rigidity and sensory disturbances.

Atrophy is said not to occur in the leg in this disease, but in this case it has occurred in the left leg. There is no doubt that the lesion was one of the spinal membranes, and to be certain of the diagnosis of pachymeningitis cervicalis hypertrophica, we must eliminate Potts' disease and tumour of the membranes.

Potts' disease is excluded by the absence of spinal tenderness, deformity, cachexia and fever, also by the absence of scrofula.

Tumour is more difficult to eliminate, but the gradual recovery excludes it, so that there can be little doubt about the case. A traumatic form of the disease has been described, and this case was probably due to injury.

The position of the hands shows the lesion to have been in the upper half of the cervical enlargement of the cord.

Reviews and Notices of Books.

Outlines of Psychology. By JAMES SULLY, M.A., &c. London, Longmans and Co., 1884. Demy 8vo., pp. 711.

An Introduction to Mental Philosophy on the Inductive Method. By J. D. MORELL, A.M., LL.D. London: Stewart and Co. Crown 8vo., pp. 390.

THERE is not much in this book that calls for notice in a Journal of Neurology. The problems of subjective psychology as distinguished from philosophy are treated fully and clearly, but that aspect of the subject which deals with nervous processes, and with the relation of these processes to the operations of mind, does not occupy a prominent position in the dissertation. Indeed, considering that the work is written with special reference to the theory of education, it is probable that its value from this point of view would have been enhanced by a more copious and more formal inclusion of the neurological side of the question. Education, as Mr. Sully insists with much force, is no longer believed to consist in merely stuffing the memory with words. The so-called education which begins with learning the Latin grammar by rote, and ends with making Latin verses by rule, is now recognised to be as unscientific as it is disgusting; and even some schoolmasters admit that the aim of education is not so much the acquirement of knowledge, as the training of faculty. Now the training of faculty is, on its physiological side, the development of the nervous system in certain directions. Great as is the importance of the purely mental or subjective aspect of the development, it is not intrinsically greater than that of the physiological aspect; and in view of the narrow foothold that it has in pedagogics, the need of a full and careful exposition of the physiology of the subject in a psychological work, written with special reference to education, is the greater. In treating of attention, Mr. Sully points out how "when once the fresh interest of a thing is exhausted, a further fixing of attention costs more and more effort; the mind soon wearies, and attention flags in spite of the utmost effort." From the introspective point of view this statement is complete; but its interest and its value would be greatly enhanced if the writer went on to give the physiological basis of this fatigue, increased effort and final failure. When it is explained that fatigue, partial or general, is the feeling that accompanies excessive expenditure of energy by nervous regions, locally or generally,

that increased effort means further expenditure by regions whose surplus has been spent, and whose reserve is liberated only by strong stimuli; and that exhaustion means the absence of any stimulus sufficiently powerful to unlock this reserve; the flagging and failure of attention acquire fresh interest. And when it is further pointed out that an exhausted nerve region, while it can be compelled to go on expending energy by increasing the magnitude of the stimulus, yet discharges at the expense of the integrity of its structure, and that this structure is more easily so damaged in the plastic tissues of youth than in the comparatively rigid fabric of the adult nervous system, a clear and strong light is thrown upon the danger of urging children to attend for a long time together to any one kind of occupation. Given plenty of food, and plenty of sleep, children can scarcely be too fully occupied in their waking hours, but the occupation should be very frequently varied, and varied through the widest possible range of difference. It is scarcely fair to blame Mr. Sully for omitting a subject which he does not undertake to deal with, and it is quite open to him to retort upon neurologists the imperfections and shortcomings of their own science. It is quite true that we cannot yet boast of having settled, even among ourselves, the nature of the nervous process that underlies every mental state, but still, having regard to the great importance of the working of the nervous system from the educational point of view, and to the small amount of attention that has hitherto been paid to this matter by pedagogues, it is very desirable that a psychologist of Mr. Sully's eminence and weight should help to bring it into prominence.

Dr. Morell's work is a sound and useful text-book for students, adapted specially to the examinations of the University of London, and enriched with a large number of examination papers which enhance its usefulness. It is concisely and clearly written, and well adapted to the purpose in view. The neurological aspect of the subject is not dealt with, except incidentally in the opening chapters; and here it is surprising to find a positive assertion of the existence not only of "vital force," but of "force of mind," which, we are told, includes Will. "These three forces (vital force, nerve force, and mind force) stand in the closest correlation." This is a position that no neurologist can accept, and if it were of importance with reference to the remainder of the book, it would be impossible to do otherwise than condemn the latter; but so long as subjective processes alone are dealt with, the book is reliable.

CHARLES MERCIER.

On Sclerosis of the Spinal Cord, including Locomotor Ataxy and other System-diseases of the Spinal Cord. By JULIUS ALTHAUS, M.D. London: Longmans, 1885.

THE book of Dr. Althaus¹ consists mainly of a *résumé* of those facts which already were familiar to the initiated in the profession, more especially with reference to *tabes dorsalis* or locomotor ataxy. The reader versed in neurology will therefore not discover anything essentially new in it. The general practitioner, however, for whom, as the preface indicates, the book is chiefly intended, will not always be able to discover from its perusal to whose credit the discovery of certain facts is to be placed; and to him, therefore, the Author's share in it will often appear greater than is really the case. No doubt this is quite unintentional on the part of Dr. Althaus; but so many names are already quoted, that it would have been better either to abstain from any references at all, or to complete still further the list of authorities.

The anatomo-pathological part of the book is decidedly the weakest; one feels that the Author is not standing on a ground personally familiar to him. He would not otherwise commend the method of Bevan Lewis, which savours of an antiquated microscopical technique, referring, for instance, to the investigation of ganglionic cells by compressing them with the needle under the covering-glass.

The definition and conception of Spinal Sclerosis, with which the author opens this book, is very peculiar; and it is highly regrettable that it should be precisely introduced in a work intended for practitioners. It implies that in every case we have to deal with *systematised* disease of the cord, a statement which will strike the critical reader as questionable as well as novel. Dr. Althaus does not, however, experience any scruple in placing among systematic lesions *multiple sclerosis*, which has never been yet, and never will be reduced to that group.

Inaccuracies and positive errors occur in the detailed account of the anatomo-pathological conditions. The author has evidently read somewhere that in *tabes* no healthy fibres occur among the diseased tissues of the posterior roots and columns (p. 31). But whoever has ever seen for himself a section of the cord in *tabes* will have noticed that this is not the case.

The other diseases mentioned besides Locomotor Ataxy, such as Friedreich's disease and spastic paralysis, are treated far too briefly. Under the latter heading Dr. Althaus makes an original statement on the subject of the tendon-reflexes. He distinguishes three types, cerebral, spinal, muscular; the last is stated to occur in peripheral paralysis, in contradiction with all other observers, who are unanimous in recognising the entire absence of the deep reflexes in such cases.

¹ This review has been written from the author's German edition, 1881.

Amyotrophic Lateral Sclerosis is disposed of in *two* pages. We can hardly consider this space, nor that devoted to the account of multiple sclerosis, as at all sufficient.

With reference to the aetiology of tabes, Dr. Althaus declares himself to be on the side of those who recognise a causal relationship between it and syphilis, but forgets to mention the fact that in 1881, at the International Congress, he attacked this very same view. Cold and over-fatigue are also mentioned as causes.

We do not find in the account of the initial stage any new points worth noticing; but a case of labyrinthine affection, with tabiform symptoms in the legs of a transitory character, appears to us doubtful, as neither pupillary rigidity nor loss of knee-jerk are mentioned. We need not discuss the statement, that attacks of monoplegia and aphasia are not uncommon at the outset of tabes. On the whole, however, the phenomena observed in the initial stage are carefully put together and described.

With reference to the second period, dating from the appearance of ataxy, we are told that it occasionally begins with fever. It appears to us questionable whether this symptom does not really belong to some other incidental morbid state, which, by the depressed condition of the general forces, brings into prominence the ataxy which may have previously existed in a very slight degree. Among the various tests by combined movements suitable to the investigation of incipient ataxy, the Author mentions making the patients walk backwards, which is a difficult performance for many even at a very early stage.

Dr. Althaus mentions the various theories propounded to explain the ataxic phenomena. He adopts the "sensorial" hypothesis, without bringing any new arguments in its support. He applies the term "static ataxy" to describe disturbances of equilibrium; whereas Friedreich, who introduced the expression, included under it ataxic movements during apparent rest, quite independently of disturbed equilibration.

Under the heading of the differential diagnosis, diabetes mellitus is especially mentioned, which presents certain nervous symptoms not unlike those of incipient tabes. These, however, we think, can prove perplexing only when the urine is not tested for sugar. It would certainly be difficult in a case where both diseases coexisted to refer each symptom to its true origin; recent French observers have stated that the knee-jerk is absent in typical cases of diabetes. The discussion of the prognosis and therapy of tabes offers no point of special interest. Where syphilis exists, innunction of oleate of mercury is recommended. Ergotin is said to have occasionally good results. Short and weak electrical currents are supposed to be the best. Strychnia is not recommended.

In conclusion, we are bound to acknowledge, much to our regret, "that the darkness which has hitherto," viz. up to the publication of Dr. Althaus's book, "surrounded the question of tabes," has not in the least been dissipated by his exertions. Neither was it required to make us conscious of the imperfection of our know-

ledge, or inspire us with a wish to learn more on a subject which admittedly constitutes an ample field for investigation. Books which are mere compilations never serve to stimulate research.

Prof. SCHULTZE, M.D. (Heidelberg).

On the Functions of the Marginal Convolution. By E. A. SCHÄFER, F.R.S., and VICTOR HORSLEY, B.S., F.R.C.S. Reprint from the 'Proceedings of the Royal Society,' March, 1884.

THE elaborate researches of Professor Ferrier, following the very suggestive but incomplete experiments of Hitzig and Fritsch, clearly showed the fronto-parietal region of the outer convex surface of the cerebral cortex to be separable into distinct areas, which regulated the movements of very different groups of muscles.

The centres thus described and mapped out by Ferrier included those for movements of both limbs and the face and neck. The neck muscles, which were noticed to be represented on the outer surface of the hemisphere, were the rotators of the head.

Schäfer and Horsley have completed this list of motor centres by finding that the trunk muscles are also represented (as might have been imagined) by centres in the cortex cerebri, and that these centres are placed in the marginal convolution. Most experimenters had previously stated that the mesial surface of the cortex of the hemisphere was not excitable, and that at any rate it did not contain motor centres other than some similar to those on the outer surface of the hemisphere, and, further, this statement only rested upon one experiment.

The methods employed by Schäfer and Horsley were those originally employed by Ferrier, viz., stimulation of the cortex by the current obtained from the secondary coil of a Dubois Reymond induction coil, with Helmholtz' side wire, to equalise the effects of the make and break shocks, the current being applied to the brain by means of platinum electrodes, which were carefully insulated to their blunted tips and bent so as to make it possible to introduce them into the longitudinal fissure without hampering in any way the circulation in the cortex. The current employed was almost invariably that obtained from one Daniel cell, and the secondary coil was usually between 7 and 8 cm. from the zero point on the side scale of the primary coil. The current thus obtained gave a distinct but not painful sensation when applied to the tip of the tongue. The animal being anaesthetised with ether (both chloroform and morphia giving inconstant and uncertain anaesthetic effects), the marginal convolution was reached by removing from the vertex a small corresponding oblong piece

of the skull, and turning up towards the middle line a narrow flap of dura mater. The large veins which enter the superior longitudinal sinus from the outer surface of the hemisphere were carefully avoided, and the electrodes gently slipped between the falx and the mesial surface of the hemisphere.

In this way the experiment was performed with extremely little disturbance of the parts, a point of the greatest importance.

The authors discovered that only a portion of the marginal convolution is excitable, and it is very interesting to note that this area exactly corresponds in extent (antero-posteriorly) with the excitable portion of the outer surface of the hemisphere. Thus it is limited anteriorly by a line which passes along the front of the centre for the rotation of the head, or, in other words, which is opposite the function of the posterior and middle thirds of the superior frontal convolution; while, posteriorly, movements can be excited by stimulation as far back as a point opposite the centre of the parietal lobule. The posterior third of the marginal motor region consequently includes the paracentral lobule of Betz, which that observer showed to contain very numerous large pyramidal motor nerve corpuscles.

The rest of the mesial surface of the hemisphere the authors find to be inexcitable. This is of course what might have been expected of the gyrus fornicatus, seeing that there is scarcely any connection between it and the direct pyramidal tract.

The centres for the trunk muscles Schäfer and Horsley find to be arranged in the excitable portion of the marginal convolution, in an order from before back, which corresponds both with the arrangement of the muscles in the body, and also with the related centres for the limb muscles.

Thus arranged in the following order from before backwards, we find the centres for

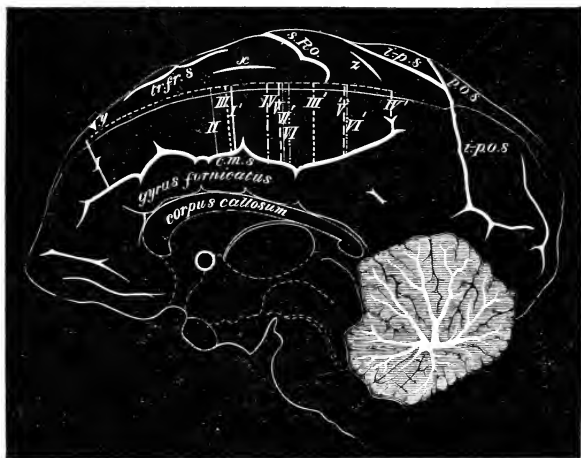
- (1.) Flexion and extension of elbow.
- (2.) Adduction of shoulder.
- (3.) Retraction of shoulder blade often combined with outward rotation of arm.

Owing no doubt to the fact that the trunk muscles are practically the servants merely of the limbs, and can be shown from daily experience rarely to move except to fulfil the requirements of the extremities, the centres for the movements of the trunk in the marginal gyrus are more complex than those centres of the extremities situated on the outer surface of the hemisphere, and therefore when excited by stronger currents, they cause perfectly co-ordinated action of the distal segments of the limbs as well as contractions of the trunk muscles.

- (4.) Arching of dorsal region of trunk (spine) with lateral flexion of spine, so that it is arched with the concavity towards the opposite side.
- (5.) Same movements in the lumbar region.
- (6.) Flexion of hip. Iliopsoas muscle.

- (7.) Extension of hip, glutæi, &c.
- (8.) Hamstrings and extensors of knee.
- (9.) Movements of ankle (flexion, extension, eversion, and inversion, &c.).
- (10.) Movements of digits.

It will thus be seen, that, as first stated, we find, even with a moderate stimulation, that groups of muscles are thrown into action which belong both to trunk and periphery. Schäfer and Horsley show, however, that in the middle of each "centre" a minimal stimulus evoked contraction often of a single muscle, and



I to I'. Motor region of shoulder and arm; II to II', of trunk muscles;—III to III', of hip muscles;—IV to IV', of thigh muscles (hamstrings chiefly);—V to V', of ankle;—VI to VI', of digits. Weak excitations in each centre produce localised effects; stronger ones, associated movements of muscles innervated by the overlapped region.

always action of only those muscles which had precisely the same effect on the movements of one joint, or, as in the particular case of the spine, a series of similar joints.

The centre of the thigh muscles is just opposite the upper end of the fissure of Rolando, and consequently occupies the site of the paracentral lobule. The leg centre consequently dips over into the median fissure a good deal more than indicated by Ferrier.

Schäfer and Horsley publish in a note an important experiment as a corollary to the above researches, in which they have removed

not only the centres on the outer surface of the hemisphere, but also their centres for the trunk muscles as well. The result is absolute hemiplegia. The paralysis of all the muscles on the opposite side of the body is apparently complete. This experiment of itself would show that the trunk muscles of one side are for the most part governed by the opposite hemisphere; and the results of stimulation show the same thing, for observation of bilateral movement following unilateral stimulation was very exceptional. Further, they have found, by microscopical examination of the lesion and spinal cord, that there are very marked descending degenerations in cases where they extirpated the excitable portion of the marginal convolution only. The bearing of this upon the explanation why the trunk muscles in hemiplegia so often escape paralysis, or are only temporarily affected, will be obvious, and Mr. Horsley published in the 'Lancet,' of July 5th, 1884, a mode of accounting for this apparent peculiarity in the light of the above-mentioned researches. An abstract of his views will be found at page 132 of the present number.

A. DE WATTEVILLE.

Histoire et Critique des progrès réalisés par la physiologie expérimentale et la méthode anatomo-clinique dans l'étude des fonctions du cerveau. Par le DR. LEVILLAIN.—Paris : Delahaye, 1884.

IN a volume of about 300 pages the Author gives us a very readable—if partial—account of the growth of our knowledge concerning the functions of the brain. As he is apparently unfamiliar with any language but the French, it is only natural that he should fail to give a proportionate share of his attention to contributions from other countries. But it is a serious blot on the book, and one which reduces its scientific value considerably, that the experiments and views of such men as Goltz and Munk are not mentioned, and that even their names do not appear in the otherwise very incomplete bibliography. Several Italian observers also ought to have received some notice at the hands of the Author. England is represented in this account by Hughlings-Jackson and Ferrier. To the former, Dr. Levillain recognises the merit of having been first to admit the existence of motor functions in the cortex. For though as early as 1827 Bravais had described cases of what is now known as cortical or Jacksonian epilepsy, he had failed to interpret the phenomena correctly. And Bright likewise, who had clearly described cases of the same nature, did not succeed in discovering that which alone gives a scientific value to clinical and pathological observation, viz. the true explanation, the law which connects the lesion with the symptom. He seems to have accounted for the existence of convulsions without loss of

consciousness by assuming that the irritation was transferred from the point of the lesion to the subjacent motor ganglia.

As the title of the volume now before us would lead one to anticipate, the Author deals first with the data of experimental physiology. After reviewing the early period (Majendie to Longet and Vulpian, 1820-1870), he divides the more recent experiments under three headings: "Method by Electric Excitations,"—"by Interstitial Injections,"—"by Limited Extirpations." The second of these methods never gave any reliable results, as might well have been expected. The third, on the other hand, since Ferrier and others introduced the antiseptic mode of operation and treatment into it, has yielded brilliant results. But the Author, who, as we have already mentioned, never alludes once to the experiments of Goltz and Munk, does not seem to be aware of the importance of "Listerism" in cerebral surgery, as practised on monkeys. In fact, it is probably owing to the neglect of due precautions in the after-treatment, and to the complications arising therefrom, that the operative procedures of Munk and others give results discrepant from those obtained in this country. Much idle talk has been expended over the first method. The "diffusion" of the electric current through the underlying tissues has been very erroneously brought forward by opponents as sufficient to explain the results obtained from the excitation of a limited area of the cortex. Any one conversant with the physical laws of electricity, and possessing sufficient practical experience of its effects as observed in the application to the organism, will have no difficulty in reducing the objections raised on that score to their just value. This technical difficulty is a purely imaginary one; moreover it is illogical; for if a sufficient diffusion did take place under the conditions of the experiments, it would not support the position assumed by the antilocalisators. It would obviously be as fatal to their position to demonstrate that the efferent fibres from certain regions of the cortex are motor, as to show that the cortical cells of those regions are electrically excitable.

The second part of Dr. Levillain's book gives a *résumé* of the labours of Charcot and his school, from the anatomo-clinical point of view. English and German contributions to the subject, including Nothnagel's classical *Topische Diagnostik*, receive but scanty recognition in the shape of a casual reference. But as it stands, this part forms an interesting chapter in the history of the question. The subject of Aphasia is touched upon, but inadequately. Those of our readers interested in it will, however, find it more exhaustively handled in the recent thesis of another pupil of the Salpêtrière, Dr. Bernard. A revolution has at last been successfully accomplished in the doctrines which have kept French observers spell-bound, as it were, since the days of Broca and Trouseau. Prof. Charcot has taken up the question, and with his characteristic force and clearness of exposition introduced to his disciples, and to the profession generally, the results reached by the psychologists of the new English school, which so admirably fit in with the recent pathology and physiology of the brain as an organ of

mind. The labours of H.-Jackson, Bastian, Broadbent, as well as Wernicke and Kussmaul and others on Aphasia, have now at last been recognised in France, where, with few exceptions, such as the thesis by Skwortzoff, the literature of the subject had remained confined within the narrow original limits.

An interesting case is described, at page 177, of sudden loss of the power of reviving the visual impression of objects (without verbal blindness). A gentleman of high intellectual culture had his visual memory developed to such a degree as to rely upon it for the greater part of his psychical operations. One morning he awoke with a feeling of confusion, and discovered that he had lost the power of revivifying past visual impressions, or recognising objects and persons he knew before. This case, as the Author remarks, is a good illustration of the distinct localisation of the various memories which form the basis of the manifestations of mind. It is from observations of this order, patiently collected and confirmed by autopsies, that our future knowledge of the higher functions of the brain has to be elicited. Not that the resources of experimentation are exhausted, or that its assistance can be dispensed with; for even where clinical investigation must, from the nature of the problems, lead the way, the discipline of comparative and physiological methods is required to act as a wholesome check upon hasty generalisations.

A. DE W.

The Brain and its Functions. By I. LUYB, M.D. Second Edition. London: Kegan Paul. pp. xviii., 327.

The Brain as an Organ of Mind. By CHARLTON BASTIAN, M.D., F.R.S. Third edition. London: Kegan Paul, 1885. pp. xviii., 708.

THE volumes before us both belong to the International series, but beyond this fact, and the similarity of their titles, have but little in common.

Of Dr. Bastian's work we need not say much here, for we trust that it is already in the hands of every reader of 'BRAIN.' Though not immediately intended for professed physiologists or medical practitioners, it constitutes a valuable contribution to the literature of the subject, and we are glad to see new editions of it rapidly succeed to one another.

We regret not to be able to say the same of Dr. Luys' book, which in our opinion is calculated to mislead and hopelessly confuse the unwary reader. The first part is anatomical, and contains an account of the author's peculiar views concerning the structure of the brain. Dr. Luys starts from the extraordinary statement, that microphotographs of brain sections afford clearer views of the structural details than the direct examination of them. As an instance of the results he obtains by his methods, we will take the optic thalamus, in which he thinks he has discovered four

sensory ganglionic centres, wherein olfactory, visual, sensitive, and auditory impressions are respectively "condensed" in their way up to the cortex. The sense of taste, it will be observed, is not represented in this arrangement, which, after the researches of Forel and others, we may well characterise with Schwalbe ('Lehrbuch der Neurologie,' p. 713) as fantastic and hopelessly erroneous.

It is difficult to understand how observers can still be found who, closing their eyes to the clearest teachings of physiology and pathology, pretend to solve the problems of cerebral function by purely anatomical data. Quite recently attempts were thus made to show that the decussation of the descending tracts took place in the corpus callosum only; and again that Broca's convolution is not a motor centre of speech, because fibres cannot be traced from it to the internal capsule. The whole of Dr. Luys' doctrine is tainted with similar tendencies. It is enough for him to find that the cerebral cortex contains layers of small and of large cells to argue that it has sensory and motor functions throughout its extent, because in the cord the motor cells are large, the sensory small. The calm assurance with which the author describes his theories is never once troubled by the consciousness, that he is in flagrant opposition to some of the best established results of modern science. He never alludes to them, nor lets his reader suspect that his opinions are shared by no other observer of note.

The second and third parts are devoted to the "general properties of the nervous elements," and to "the evolution of the processes of cerebral activity." We shall not attempt to criticise Dr. Luys' treatment of this part of the subject, but content ourselves to illustrate it by means of a quotation or two, typical of the style adopted by the author. "This form of sensibility [moral] which runs riot in spite of us, these plexuses of the *sensorium commune*, which comprehend in themselves all the diffuse sensibilities of the organism, represent, then, a sphere of nervous activity in erethism, always living, always feeling, in the bosom of which our total personality lives and vibrates. There, in this mysterious dwelling, it is in perpetual intercourse with the perpetual movement of the operations of cerebral life." "The human personality is seized upon by the arrival of the excitation arriving from the personal world. It enters into contact, and becomes associated with this; and from this intricate connection results a true intra-cerebral automatic radiation, which produces the apparition of a series of agglomerated secondary ideas. But the matter does not stop there: this inner personality having been thus seized upon, its sensibility having been touched in any manner whatever, has reacted by virtue of the vital forces that vibrate in it in a latent condition,—it has been affected in the direction of its most profound affinities, and necessarily this reactionary period betrays itself by an unconscious desire for such or such a definite object, and by a repulsion from such or such another."

Such are the "functions of the Brain," according to Dr. Luys.

A. DE W.

Les Troubles de la Parole. Par le Professeur A. KUSSMAUL. Traduction française par le Dr. A. RUEFF. (pp. 375.) Paris: J. B. BAILLIÈRE, 1884.

THE classical treatise on the diseases of Speech by Professor Kussmaul first appeared as a volume in Ziemssen's 'Cyclopædia.' English readers who do not know German and do not have access to the American translation of the series, have hitherto been debarred from the perusal of the work before us, the only one of its kind. It will therefore be welcome news to many that Kussmaul's monograph has been placed within their reach by the careful translation into French which Dr. Rueff has lately supplied. We need not go into any details concerning the method adopted by the Author in the treatment of the difficult subject of Language. His teaching has been generally recognised as sound as well as exhaustive; and his views have been generally adopted by German writers. Those of his readers, however, who are conversant with the results of this English school of psychology will notice the disadvantage at which the Author stands in the introductory chapters, from his inability to shake off the semi-metaphysical standpoint still current among many of his countrymen. The discussion on aphasia, and of articulatory disturbances, which forms the bulk of the work, is full of facts and rich in suggestive thoughts.

A. DE W.

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1. *Rivista di Filosofia Scientifica.* Diretta da E. MORSELLI.
 2. *L'Origine dei fenomeni psichici.* Di G. SERGI. Fratelli Dumolard, Editori. Milan and Turin, 1885.

THE political resurrection of Italy has been speedily followed by a scientific renaissance among its people. On every side signs of increasing activity become manifest. We have now before us some numbers of a new 'Review of Scientific Philosophy,' which is just entering upon its fourth year of existence, and which is conducted on much the same lines as the well-known 'Revue Philosophique,' edited by M. Ribot. Positive psychology and neurology are amply represented in its pages. Thus we may notice an interesting "Study of Experimental Psychology: on the Time of Perception of Colours," by Buccola and Bordoni; articles on "The Notion of the Individual in Biology," by Bonelli; and "Researches on the Influence of Hypnotics on the Duration of Psychical Acts," by Cervello and Coppola. Professor Fano gives us a discourse on "Physiology as an Autonomous Science;" and finally, Sergi publishes a chapter, "On Psychical Acts as Functions of the Organism," from his new work since published.—'L'Origine dei Fenomeni Psichici e loro significazione biologica,' is a title

sufficiently descriptive of the tendency of the book, which takes its standpoint on the modern monistic hypothesis. It is pervaded with the true Darwinian and Spencerian spirit, and the author fearlessly deduces his conclusions, driving them to their uttermost consequences. Consciousness arises and is developed in the series of animal life, as "a function of protection." With a great fulness of detail, the Author unfolds to the reader the ascending steps by which the evolution of mind is accomplished according to the principles of the struggle for life.

This new work by the Professor of Anthropology in the University of Rome forms a volume of the "International Series," and will therefore, very probably, become accessible to the bulk of English readers at no distant date.

A. DE W.

Zur Einleitung in die Elektrotherapie. Von Dr. C. W. MÜLLER. J. F. Bergmann, 1885. 1 vol. 8vo. (pp. 187).

THIS little book embodies the results of most praiseworthy efforts in the scientific direction fortunately taken by Electrotherapeutics of late years. The Author takes the same standpoint which we ourselves have always assumed, viz. that the fundamental question in electric treatment is one of physics, and not of physiology. It is with much satisfaction that we see the question of current measurement occupy a more and more prominent position in the various papers and books published on this subject, and the unit we first proposed (milliweber, or milliampere) adopted by all good observers. The work now before us consists of two parts. In the first the Author furnishes us some very precise data concerning the various galvanometers intended for medical use; he comes to the conclusion that the new astatic vertical instrument of Hirschmann is the best. The second part is much more practical, and deals with the question of the density of the current in actual applications to the human bodies. By density is meant the ratio between the strength of the current, and the surface to which it is applied. This surface is obviously equal to that of the electrode. Hence the expressions $\frac{1}{5}$, $\frac{1}{10}$, $\frac{2}{15}$, $\frac{2}{24}$, and the like, indicate that current of 1 or 2 milliamperes is applied by means of an electrode of 5, 10, 15, 24 square millimetres. The time during which the current is allowed to flow is also indicated, and thus a very accurate system of dosage is obtained. The Author's experience leads him to formulate the aphorism that electric applications should be weak, short, and frequent. Indeed, in his practice he reminds one of the homœopath and infinitesimal doses. We must confess that we have never tried such a plan, for the simple reason that we have found a more decided course of action generally useful, especially in cases of organic disease. To give an instance; instead of a current of $\frac{1}{15}$ for one minute daily, as recommended by Dr. Müller in sciatica, we have often found great and almost immediate relief from

applications of currents as strong as the patient can bear, with very large electrodes ($\frac{2}{15}$ or more), for 10 to 20 minutes, with a moderate faradic current flowing at the same time. In all except very chronic cases this strong application is to be repeated more than once a day.

We must, however, recommend Dr. Müller's little book as a thoroughly exact, honest, and painstaking performance. He writes obviously with enthusiasm, and for the instruction of the reader rather than for his own interest or glorification. He is at the same time scientific and practical, and the spirit he displays stands in refreshing contrast with the tendencies displayed by too many writers on electrotherapeutics.

A. DE W.

A Practical Treatise on Disease in Children. By EUSTACE SMITH, M.D. London: Churchill, 1884. 8vo. pp. 840.

THIS volume is certain to become popular among medical practitioners, for whom it is intended from the essentially practical standpoint assumed throughout by the Author. On every page we discover evidences of its origin; not the outcome of painstaking compilation of authorities, but the natural flow from a mind well stocked with personal experiences. Its function is to serve as a guide at the bedside, not as a didactic teacher at the reading-desk; and so the subjects of diagnosis and treatment have received the greatest share of the Author's attention.

Dr. Smith did well to alter the customary "Diseases of Children," into "Disease in Children." There are no diseases which deserve the former name; but, on the other hand, the physiological characteristics of childhood which modify disease in many ways, and the peculiar difficulties of diagnosis and special requirements of treatment in the case of patients in the first period of life, fully justify the distinction implied in the latter.

This remark applies more particularly to nervous disorders, to which 140 out of a total of 800 pages are devoted in the work before us. The nerve-centres form the most differentiated structures of the body and are the last to reach their full differentiation. The new-born child is an automaton, and his psychical existence a mere manifestation of vegetative reflexes; under the influence of the various external stimuli, the rapidly growing hemispheres are the seat of extraordinarily active processes, and the motor tract is subject to ceaseless demands from the higher centres. Inhibition is still in abeyance, and there is no localisation of single excitations, which, diffusing themselves over the whole cortical surface, give rise to disproportional external effect. It is this diffusibility of nervous processes which led psychologists to speak of the "spontaneity" of the nerve-centres, and gives a peculiar aspect to many of the morbid phenomena of childhood.

The immature condition of the higher centres, and the greater

activities of the vegetative functions in early life, would have led us to expect *à priori* that the treatment should differ from that adopted for the adult, not only quantitatively, but qualitatively. Nothing can be more fallacious than the mathematical dosage of remedies adopted in the usual textbook of *Materia Medica*, when no account is held of any circumstance except of age. Dr. Smith has fortunately quite emancipated himself from the orthodox superstition; and we are glad to see him recommend, for instance, arsenic in 10-drop doses or more in chorea, where our experience agrees with his as to the value of treatment, if heroic enough. Tincture of belladonna, again, may be given beneficially in doses of thirty drops to infants, upon whose immature brains the drug cannot have any toxic effect, whilst such quantities exert a distinct influence upon the lower reflex processes. The author's directions as to the hygienic and dietetic management of children are full and practical; and the whole question of treatment is handled with a mastery which only years of thoughtful experience and observation can impart.

A. DE W.

Lectures on Mental Disease. By W. H. O. SANKEY, M.D.—
2nd Ed.—London: Lewis, 1884.

WE welcome with pleasure the second edition of Dr. Sankey's '*Lectures on Mental Diseases.*' Few men in this country have had a wider or more varied experience of insanity than Dr. Sankey, and no one perhaps has a greater right to speak with authority on the subject than himself. Excepting the first 99 pages, devoted to mental science or physiology, the book is eminently practical. The first part, notwithstanding the very excellent plates of microscopical structures which illustrate it, might, we are inclined to think, have been omitted, or at any rate much curtailed, with advantage. The remainder and by far the larger part of the book is all good. Dr. Sankey prefers to include all forms of insanity, excepting General Paresis and Idiocy, under the head of Ordinary Insanity, on the ground that they are all phases or stages of the same disease. In this view he is probably right. Nevertheless, under this head he describes the generally recognised varieties of the disease just as most other writers do. And considering that writers generally would agree with him in opinion, and that their separate groups are made, as are his separate descriptions, mainly for convenience of arrangement, the difference between him and them seems apparent rather than real. At any rate it is not very important. We do not say this with the object of fault-finding; and indeed are disposed to admit that there may be some advantage in impressing upon students the fact of the essential identity of most varieties of insanity. The chapters on treatment, and on the legal relations of insanity, are full and satisfactory.

J. S. BRISTOWE, M.D.

An Introduction to the Study of the Diseases of the Nervous System. By THOMAS GRAINGER STEWART, M.D., F.R.C.P.E., &c. Edinburgh; Bell and Bradfute, 1884.
Diseases of the Spinal Cord. By BYROM BRAMWELL, M.D.—Second Edition. Edinburgh: Pentland, 1884.

THE clinical study of nerve disease and disorder has come to be more and more arduous with advancing knowledge; and it is doubtless true that some guide is requisite for those who are beginning to investigate such cases, and to search for causes among the multifarious abnormalities which come under their observation. There is certainly room for such a book as that before us, which tells the student what to look for in cases of supposed nervous disease, and how to look for it. It is well, especially in this department of medicine, that a clear idea should be gained of what the symptoms are which point to nerve disease, and what the known or probable causes of such symptoms may be, before the so-called "diseases" of an established nosology are each of them separately studied. In such a book as this, originality is of course not to be looked for, nor would it be, indeed, desirable; and it is equally impossible in the space of the volume for acknowledgment to be made of the various thoughts and illustrations taken from other authors. It is in the main a compilation. But Professor Stewart has done well the work he has undertaken, especially in the part relating to symptomatology. The first three lectures on the medical anatomy of the nervous system would be of greater value as lectures, clearly illustrated, than they are for perusal by the reader; one at least of the diagrams is unintelligible in its minuteness. The altered symptoms, however, occurring in disorder of the various departments of the nervous system are very clearly set forth, and the description is frequently accompanied by excellent plates. The book, as a whole, is carefully written; and will doubtless be widely used by those to whom ampler volumes are inaccessible.

Dr. Bramwell's book well deserves the success it has already met with. Translations into German, French and Russian, and a second English edition reached within two years and a half of its first publication, show how keenly it has been appreciated by the students and practitioners for whom it is intended. The characteristic feature of the work is the fulness and perfection attained in the illustrations. In this edition the number of the beautiful chromolithographs and drawings representing the normal and pathological structure of the cord has been still further enlarged; and a chapter added on "Concussion;" whilst the text bears the evidence of careful revision throughout.

H. B. DONKIN, M.D.

Critical Digest.

ON THE COMBINATION OF LATERAL AND POSTERIOR SCLEROSIS IN THE SPINAL CORD.

BY J. A. ORMEROD, M.D.

IN the classification of chronic degenerative diseases of the cord, a distinction is generally admitted between those that are "systematic" and those that are "diffuse;" the systematic diseases being those that affect only certain definite physiological tracts, and spread only in the direction of the affected tracts, the diffuse being indiscriminate in their onset and mode of extension. The division of the cord into such physiological tracts has been based on the following methods: (1) observing the march of degeneration in fibres separated from their nutritive centres, [secondary degeneration: method of Türck and Waller], (2) observing the different times at which different sets of fibres reach their full development [method of Flechsig]. A striking agreement obtains in the results of these two methods; which is rendered still more striking by the fact, that (3) certain chronic diseases of the cord whose precise causation is unknown (primary degenerations) map out, at least in their simpler forms, corresponding regions of the cord. These are the "systematic" diseases, or as they may be more shortly called, tract-diseases.

The first and second of these methods have been principally applied to the white substance; and we shall in this article consider disease of the white substance only, though certainly the selective power of disease is manifested also in the gray. But under our heading will be included at least two varieties of disease—one in which the sclerosis of each column coincides with the known physiological tracts contained therein (for this we propose the name multiple tract disease); the other, in which the sclerosis of one or other column (generally it would appear the lateral column) does not coincide with known tracts, though too regular in its distribution to be called in the strictest sense diffuse.¹

We shall first give a brief abstract of such cases as we have been able to find in foreign literature.²

¹ It may be noticed that Westphal ('Arch. für Psychiatrie,' etc., vol. viii.) gives a case of posterior sclerosis combined with disseminated sclerosis of other parts.

² We have unfortunately been unable to obtain the account of a case by Mr. L. Hamilton, 'New York Medical Record,' 1879.

(1.) PIERRET. 'Arch. de Physiol,' vol. iv., 1871-1872 :—

A female, æt. 60. Complete paralysis of the lower limbs for twelve years; which had been preceded by weakness and pain in them. Sensibility absent in the legs: arms thin, and their movements inco-ordinate. State of torpor.

Post-mortem.—Degeneration of the whole of the *posterior* columns and atrophy of the posterior nerve-roots. *Lateral* columns, posterior parts diseased, chiefly in the lumbar region.

[This may have been simply an example of the transverse spreading of the sclerosis from the posterior columns to the lateral. It is, however, quoted by Westphal in his article, to which we shall presently refer.]

(2.) PREVOST. 'Arch. de Physiol,' series ii. vol. iv. 1877.

Male, æt. 51. Duration of disease, between seven and ten years. Initial symptoms, pains in loins, weakness in legs and in right arm, irritation in skin of right arm, difficulties in defæcation. Then increase of weakness till patient could not walk or sit up, double vision, progressive amaurosis. While under observation the lower limbs were almost paralysed, the upper limbs ataxic, sensibility diminished. Reflex movements (to pinching, tickling, &c.) were very violent, especially on the right side. Finally, emaciation set in: death from bedsores.

Post-mortem.—There was in the *posterior* columns sclerosis, of an extensive area, throughout the whole length of the cord, but most extensive in the dorsal region. In the *lateral* columns, sclerosis, also most marked in the dorsal region, and diminishing gradually as the lumbar and cervical regions were approached. It involved chiefly the posterior part of the lateral columns, but extended forwards along their periphery. In the dorsal region, it dipped deeply into the substance of the cord. Except in a few sections the lateral sclerosis was not continuous with that of the posterior columns, and the author insists that it could not be regarded as a simple extension of the latter.

The violence of the reflex movements (the skin-reflexes only are alluded to) led him to conjecture, during life, the existence either of a transverse myelitis or a lesion of the lateral columns.

(3.) KAHLER AND PICK. 'Archiv für Psych.,' vol. viii. 1878.

A case clinically resembling the hereditary ataxia of Friedreich, remarkable *post-mortem* for the exactness with which the posterior columns, cerebellar tracts, and pyramidal tracts were picked out by disease. Of this case we have already given an abstract in BRAIN, No. xxv., p. 114.

WESTPHAL. 'Archiv für Psychiatrie,' &c., vols. viii. and ix., 1878-1879, gives a series of five cases :—

(4.) Female, æt. 45. Duration, nine years in all. Disease began with pains in back and in left leg, then numbness in fingers, shooting pains in upper and lower limbs, double vision. In five years' time difficulties of micturition, and of defæcation, and of

walking began. While under observation (a period of three years) there was noted—paralysis and impairment of sensibility in the lower limbs, ataxy and impairment of sensibility in the upper limbs; paralysis of the bladder, paralysis in the districts of the right third and sixth nerves, and of both trigeminal nerves; affection of the cornea, amblyopia, ataxy of the facial muscles, attacks of dyspnoea and delirium: general emaciation, especially in the legs.

Post-mortem.—The following nervous lesions were found:—

Chronic lepto-meningitis: degeneration of the posterior nerve-roots. Within the cord the *posterior* columns were sclerosed, over nearly their whole area, through the whole length of the cord. There was also degeneration of the *lateral* columns (differing somewhat in its microscopical characters from that of the posterior) which occupied, in the lumbar and dorsal regions, an area at the posterior part of the columns not reaching to the periphery of the cord (crossed pyramidal tracts?). In the cervical region this area was healthy, but here at the periphery, in the middle part of the columns, there was a thin zone of disease, faintly marked and only to be made out with the microscope.

Further there was degeneration of the ascending root of the trigeminus, reaching upwards to the decussation of the pyramids: atrophy of the trigemini and partial atrophy of both optic nerves.

(5.) Female, æt. 51. Nineteen years before her admission she had noticed, during pregnancy, a numbness of the abdomen; next her gait became waddling. After an abortion, weakness of the right leg appeared: then a dislocation of the hip joint in consequence of joint-disease. [The author does not consider the joint disease to be of the special form described by Charcot.] Five years before admission there appeared numbness and weakness of the upper limbs, and four months before admission inability to walk, difficulty of micturition and defæcation, double vision, impairment of speech, asthmatic attacks. After admission, there was observed complete paralysis of the lower limbs, paresis of the upper limbs, impairment of sensibility in both upper and lower: no rigidity. There were nervous disturbances in the districts of both trigemini, also increased flow of saliva, congestion of the face, sweats, and vomiting. Later still, impairment of speech and of swallowing, general emaciation, death.

Post-mortem.—There was chronic lepto-meningitis; and in the cord, sclerosis universal or nearly so of the *posterior* columns. In the rest of the cord there was disease reaching from the anterior fissure to the tip of the posterior cornu, involving an area which varied at different levels, as follows: in the cervical region there was only a thin zone of disease reaching along the periphery and along the anterior border of the posterior cornua; but in the dorsal and lumbar regions this zone became broader, and extended more and more deeply into the substance of the lateral columns. There was also some disease of the gray substance of the anterior cornu, most marked in the lumbar region.

(6.) Female, æt. 45. Duration of disease, six years. It began with bouts of pain at the left lower ribs, and vomiting; next there developed gradually weakness and anomalies of sensation in the legs and in one or two fingers; awkwardness in using the hands, transitory diplopia, progressive amaurosis, numbness of the left face, and left side of buccal cavity. While under observation (a period of two years), the weakness gradually increased, first in the legs, then in the arms (the left especially); the gait, at first somewhat ataxic, became paretic in character; the movements of the legs, examined while the patient was lying down, were ataxic, but finally evinced much loss of voluntary power. The legs, however, were subject to involuntary movements. In the left arm there was ataxy and loss of power; in the right, slight ataxy. The impairment of sensibility in the upper and lower limbs gradually increased and spread to the abdomen. Girdle-sensation present; skin reflexes normal; knee and foot phenomena absent; no paralysis of bladder; impairment of sensibility in the district of both trigemini; flushings of the face and neck; sweating; frequent pulse; painful vomiting; much general emaciation.

Post-mortem.—There was pulmonary and intestinal phthisis; and in connection with the nervous centres, chronic pachymeningitis of the convexity of the brain; adhesions of the spinal dura and pia-mater, especially on the posterior aspect of the cord. Within the cord, sclerosis tolerably universal of the *posterior* columns, and also of a zone at the periphery of the *lateral* columns, but this was nowhere so broad as in the last case. There was also atrophy of the ascending roots of the trigemini, of the trigeminal, optic, and olfactory (?) nerves.

(7.) Male, æt. 45. Duration of disease, four or five years. First, stiffness in the legs, girdle-sensation, transient pains, of a tearing character, in the legs; then, after an injury to the knee-joint, numbness and pricking in the feet, difficulty in walking, slight troubles of micturition and defæcation. While under observation, there was paresis of the lower limbs, stiffness of the joints, spastic gait, foot-clonus; sensory troubles in the upper limbs. The patient had chronic lung disease; diabetes mellitus supervened, an abscess in the rectum formed, and death followed.

Post-mortem.—Fluid was found in the sac of the dura-mater; adhesion between the dura and pia. There was disease both of the lateral and posterior columns, the distribution of it being much as follows. Its area was most extensive in the upper dorsal region; the *posterior* degeneration here affected both the columns of Goll and of Burdach, higher up the columns of Goll only, lower down it disappeared altogether. The *lateral* degeneration in the dorsal region extended inwards from the periphery over a considerable area of the posterior and middle part of the lateral columns; above the dorsal region, only over a thin zone at their periphery, while below the dorsal region it became more and more limited to the area of the crossed pyramidal tracts.

Thus the distribution of the disease above and below the dorsal

region corresponded to that of a secondary degeneration proceeding from a transverse lesion of the middle part of the cord (columns of Goll and cerebellar tracts above, pyramidal tracts below), and this explanation of the facts is advanced by some recent writers.¹ Westphal himself thinks that, in the absence of a distinct patch of transverse myelitis, the disease must be considered to be primary; it corresponds to what he has observed in paralytic dementia. Clinically the spastic gait and exaggeration of tendon reflexes were so marked, that had it not been for the slight sensory symptoms and the disturbances of micturition, the case might have been taken for pure and simple lateral sclerosis.

(8.) Male, æt. 44. Duration of disease, six years; death from peritonitis, caused by ulceration round a gall-stone.

Pain in the back, transient numbness of thorax, transient diplopia and weakness of the bladder, diminished sensibility in left upper limb. Three years later, difficulty of walking and sense of constriction at the thorax. After admission, increased disturbances of sensibility, impairment of muscular sense, retarded sense of pain. Ataxy of all four limbs, and involuntary movements of them. Double amaurosis, nystagmus. No rigidity, absence of tendon reflexes.

Post-mortem.—Chronic spinal lepto-meningitis; of the *posterior* columns, tolerably universal degeneration; of the *lateral*, degeneration which in the cervical and dorsal regions was asymmetrical, involving on the left side the middle part of the column near its periphery, on the right side its posterior part and more deeply. In the lumbar region and below, the lateral degeneration became more symmetrical, and its area corresponded more nearly with that of the crossed pyramidal tracts.

Westphal's remarks upon these cases are important; and though we shall have occasion to recur to them later, we may give the gist of them here. The posterior sclerosis was undoubtedly systematic, involving as it did known physiological tracts; but the disease of the lateral columns (in some parts at any rate) corresponded neither to the area of the pyramidal tracts, nor of the cerebellar tracts, nor to a combination of the two. The relation of the disease in the lateral and posterior columns respectively is difficult to determine. Westphal's view, if we understand him rightly, is that both are primary and independent of each other, and that the irregularity in the distribution of the lateral degeneration is more apparent than real, for fibres in different tracts may, he thinks, derive their nutrition from an identical source, and thus fall victims to one and the same primary degeneration. He observed in several of the cases a difference between the histology of the lesions in the two parts of the cord; but he regards the process as essentially the same in both, the posterior sclerosis being however of older date than the lateral. Clinically the diagnosis has to be made between simple disease of the posterior columns and the

¹ MM. Ballet et Minor, 'Arch. de Neurologie,' No. 19.

combined disease: and the chief point of distinction is that, in the latter, symptoms of motor weakness are added to the ataxy. The gait becomes paretic, there is dragging rather than jactitation of the legs, and at a later stage, when the patient is examined lying down, feebleness of movement can be recognised as well as inco-ordination, though the identification of these phenomena may doubtless be difficult. At any rate, the loss of power actually observed by him is explained most naturally by reference to the lesion of the lateral columns (though the explanation has its difficulties).

Rigidity and spastic phenomena were absent, except in his fourth case (No. 7), where the lumbar part of the posterior columns was normal. Hence Westphal comes to the conclusion, "that in combined disease of the posterior and lateral columns rigidity and spastic contractures do not appear, if the disease of the posterior columns reaches down to the lumbar region and involves that part of the posterior columns known as the posterior root-zone."

(9.) RABASIU. Virchow's 'Archiv,' vol. 76 (1879), p. 74. Male, æt. 44. Duration of disease, three years. It began with a feeling of weakness and heaviness in the legs, difficulty in walking, stiffness in the legs. Then pains in the lower limbs and genitals; for which, two years after the onset of the disease, the patient took to bed, where he remained for the rest of his life. Double optic atrophy began about six months before his death. The lower limbs were observed to be rigid, the feet in the position of equinovarus. There was occasional clonic twitching of the legs. Yet the tendon-reflexes were but slightly exaggerated. There was slight diminution of sensibility in the feet. Paralysis of bladder and vesical sphincter. Death from bed sore and gangrene of bladder.

Post-mortem.—The spinal meninges were thickened and pigmented. The spinal degeneration was most marked in the dorsal region. Here in the *lateral* columns it reached from the posterior nearly to the anterior cornua and round the periphery of the anterior columns to the anterior fissure. Above and below the dorsal region the disease of the lateral columns diminished in extent. In the *posterior* columns there was degeneration, through the whole length of the cord, of Goll's tracts, and of the peripheral part of the cord just external to Goll's tracts. The histological appearances resembled those of insular sclerosis, viz., thickening of the interstitial tissue, increase of neuroglia nuclei, presence of amyloid bodies, hypertrophy of axis cylinders. The cells of the anterior cornua and of Clarke's columns were normal.

The intensity of the disease was much greater in the lateral than in the posterior columns, and with this fact the author connects the spastic symptoms observed during life. From the combination of these spastic symptoms with the sensory symptoms, bladder trouble, and optic atrophy, the diagnosis of combined lateral and posterior sclerosis had been made.

(10.) KAHLER AND PICK, 'Archiv für Psychiatric,' vol. x. 1880,

give an account of a post-mortem (without clinical history), in which they found *posterior sclerosis* limited to Goll's tracts in the upper part of the cord, and involving the posterior radicular zones lower down; *lateral sclerosis* involving the whole length of the pyramidal tracts, and also the periphery of the lateral and anterior columns. This peripheral sclerosis was, in their opinion, partly due to systematic disease of the cerebellar tracts; for there was a paucity of cells in the columns of Clarke; the cells which constitute, as they think, the nutritive centres for these tracts.

SCHULTZE, 'Virchow's Archiv,' vol. 79, 1880, gives two cases.

(11.) The first constituted, during life, one of the cases upon which Friedreich founded his original description of hereditary ataxia. An abstract of the case will be found in 'BRAIN,' No. xxv. pp. 115-120.

(12.) In the second there was no hereditary history. The patient was a middle-aged man, whose complaint had lasted ten years at the time of his death. First symptom, tearing pain in left leg; in two years' time, ataxy of legs; soon afterwards, optic atrophy, which caused complete blindness in two years more. On admission, paralysis of lower limbs with rigidity (flexion of knees and thighs, with adduction of thighs); absence of patellar tendon-reflex and of ankle-clonus; loss of sensation, except for heat, in the lower limbs. Upper limbs normal. Frequent pains in legs and back. Incontinence of fæces.

Examination of the cord shewed "fibrillar degeneration" of the *posterior* columns in their whole length; and also of the posterior part of the lateral columns. The disease in the *lateral* columns involved not only the pyramidal tracts, but also a zone at the periphery of the middle part of the columns. This peripheral zone corresponded to the area of the cerebellar tract in the cervical region, but in the dorsal region spread beyond it.

There was slight thickening of the pia-mater. The posterior roots were thinned. The grey-matter of the posterior cornua was slightly diseased. The cells of Clarke's columns were atrophied.

Apropos of this particular class of case, Schultze makes the apposite remark, that the disease is too regular in distribution for insular sclerosis, too irregular for genuine tract-disease.

Three remarkable cases are given by STRUMPELL, 'Archiv für Psychiatrie,' vol. xi. 1881.

(13.) The symptoms resembled those of amyotrophic lateral sclerosis.

Female, æt. 32. Duration of disease, three to four years. It began, after a fall on the back of the head, with feverishness, pain in the head, stiffness of the neck, slight weakness in the right leg. Three months later, decided weakness in the legs; later, slight twitching of them, slight paræsthesiæ, some difficulty of micturition. She was admitted to Hospital a year before her death; there was then considerable paralysis of the legs, with

stiffness and increased tendon reflexes; associated movements of the two legs. Sensibility normal. Slight involuntary (choreic?) movements of the arms. Some inequality of pupils. For a time there was slight improvement under galvanism, but she finally got worse. Wasting of some muscles in the legs and hands was noted; finally, incontinence of urine appeared, and death followed.

Post-mortem.—Strumpell found disease of the pyramidal tracts, and of some sets of fibres in the posterior columns, with slight disease of the cerebellar tracts. The anterior cornua were normal, but the cells of Clarke's columns were atrophied.

There was no meningeal affection.

The affection of the *pyramidal tracts* could be characterised as strictly systematic; both because the area of disease corresponded exactly with that of the crossed pyramidal tracts, and because one direct pyramidal tract was also affected. But it was not secondary to any intracranial lesion, for the pyramids of the medulla and the crura cerebri were normal. It was therefore primary, and propagated, the author thinks, from below upwards.

With respect to the *posterior columns*, although the diseased areas did not correspond exactly with what we know of the physiological tracts in this part of the cord, yet from its bilateral symmetry, and from the length of cord over which the posterior sclerosis extended, Strumpell thinks that it must be classed as systematic, as the lateral sclerosis undoubtedly was.

Roughly speaking, Goll's columns were diseased from the upper lumbar to the cervical region. Besides this, there was in the dorsal region a triangular area of degeneration, with its base at the periphery of the cord, between Goll's columns and the posterior root zones. In the upper lumbar region the posterior root zones, and in the lower lumbar region the whole posterior columns were free from disease. Thus the spastic symptoms and excess of tendon-reflexes can be accounted for in accordance with Westphal's dictum. (The spastic symptoms consisted in intermittent painful contractions rather than in permanent rigidity.)

In the wasted muscle there was no distinct reaction of degeneration during life, nor was degenerative atrophy found post-mortem: neither were the anterior cornual cells diseased. Hence the resemblance to amyotrophic lateral sclerosis was more apparent than real.

(14.) Paralysis and contraction of lower limbs, with impairment of sensibility. Exaggeration of tendon-reflexes: incontinence of urine. The patient was an old and pauper woman; the disease lasted about nine months.

The posterior columns were normal in the lower lumbar region; in the upper lumbar region traces of disease began to show themselves in Goll's columns; further up, the disease became more marked in Goll's columns. In the cervical region certain other portions of the remainder of the posterior columns were also degenerated. In the lateral columns there was well-marked degeneration of the cerebellar tracts, degeneration also of the

crossed pyramidal tracts, less intense than in the cerebellar, but becoming more marked in the lower part of the cord. The gray substance was normal, with the exception of Clarke's columns.

There was, therefore, in these two cases, simultaneous degeneration of those three tracts in the cord which contain long fibres, viz., Goll's tracts, the cerebellar tracts, and the pyramidal tracts. It is remarkable that the degeneration of the pyramidal tract was (in the author's opinion) ascending, while that of the posterior columns was more marked above than below. The areas of disease in the posterior columns were somewhat peculiar: but for details of them we must refer to the original paper.

(15.) Typical tabes, with subsequent paralysis of the lower and paresis of the upper limbs.

Male, æt. 50. Duration of disease, 19 years. First, tearing pain in the legs, and sweating of the feet: in two years' time, difficulty of walking. Blindness for 14 years, deafness for four years. On admission, there was complete paraplegia, the tendon-reflexes were absent, the muscles flaccid, their electro-contractility diminished. There was emaciation, but no fibrillar twitching of the muscles. The patient was absolutely blind, absolutely deaf, and anæsthetic over the greater part of his limbs and trunk. He had to be questioned by tracing out letters on his forehead. Incontinence of urine, cystitis, and a bed sore ended his life.

Post-mortem.—There was almost complete degeneration of the posterior columns and posterior nerve-roots, of the cerebellar tracts and (in the lower dorsal and lumbar regions) of the crossed pyramidal tracts. Also chronic meningitis, and atrophy of the optic and acoustic nerves.

(16.) SIÖLL. 'Archiv für Psychiatrie,' vol. xi. 1881.

In this case the gray matter was more extensively diseased than in any we have yet considered: there was, moreover, dementia.

Male, æt. 39. Severe fall, followed by coma and transitory paralysis. Slight weakness of the legs remained, which in five years' time began to increase steadily; sensory affections of the legs also began. On admission, there was typical chronic dementia; almost complete paralysis, with wasting of the legs: hyperæsthesia of the legs. Absence of knee-phenomenon. Weakness of the arms. Catarrh of the bladder: death from pneumonia, within a year.

The changes in the cord and medulla were as follows:—

Posterior columns.—Degeneration of the nuclei of the funiculi graciles, and of these funiculi down to the level of the last cervical nerve, with irregular encroachment upon Burdach's columns. In the dorsal region, the disease passed over from Goll's columns to the parts just external to them, in which area it could be traced, though faintly, down to the sacral cord.

Lateral and anterior columns.—Intense degeneration of the cerebellar tracts from lumbar region to medulla. Disease of crossed

pyramidal tracts from sacral to lower cervical region, and of direct pyramidal tracts from lumbar region up to second cervical nerve.

In the upper dorsal region the disease inclined to spread diffusely over other tracts of the cord. In the gray matter, the cells of Clarke's columns had disappeared; also the median group of the anterior cornual cells: in the dorsal region indeed the whole anterior cornu was diseased.

(17.) RAYMOND. 'Arch. de Physiologie,' vol. x. 1882.

Female, æt. 78.

From age of 40, pains in the legs recurring from time to time. No other symptom till seven years before her death, when walking became difficult. She took to crutches two years later. About two years before death, after more violent pain than usual, stiffness began, first in one leg, then in the other. Soon the arms were similarly affected. While under observation, there was almost complete paralysis, with rigidity of all four limbs: exaggeration of superficial and tendon reflexes: vasomotor paralysis in the arms. There was wasting of many muscles; but their electrical reactions were normal. Death from pneumonia.

There was posterior and lateral sclerosis most marked in the cervical region. The *posterior sclerosis* was limited in the cervical and dorsal regions to the columns of Goll; in the lower dorsal region to the "Bandelettes externes," and in the lumbar region had almost disappeared. The *lateral sclerosis* was also most marked in the upper cord, and upon the left side (the left limbs had been affected before the right). It covered, according to the author's plates, a fairly well-defined triangular area in the postero-lateral columns, whose base reached the periphery [= crossed pyramidal tracts and cerebellar tracts?] The number of cells in the anterior cornua and in the columns of Clarke were diminished.

In this case, therefore, as in Strumpell's two first (Nos. 13 and 14), the spastic symptoms were predominant, and the posterior sclerosis spared the lower part of the cord.

(18.) STADELMANN. 'Deutsch. Archiv für Klinische Medicin,' vol. xxxiii. part 2, p. 125.¹

Female, duration of disease, four years. Sacral pain, weakness of legs. Four weeks before admission, paraplegia, diminution of sensibility in the legs, paralysis of the bladder. On admission, state of hebetude, ptosis of right eye, paresis of left facial. In legs, absence of tendon-reflex, paresis of motion and sensation, pains. Arms not paralysed but hyperæsthetic.

Post-mortem. The cord was seen to be studded with small spots, the size of a pin's head, apparently hæmorrhagic.

The *posterior columns* were affected thus: in the cervical region

¹ The account given here is taken from an abstract in the 'Neurologisches Centralblatt,' 1883, p. 327.

Goll's columns were diseased; somewhat lower, Burdach's columns became involved; and lower still, the whole posterior columns were diseased. As to the *lateral columns*: in the upper cervical region there was a zone of degeneration at the periphery, which was broadest at that part where the cerebellar tract is usually placed; at the level of the cervical swelling, this peripheral degeneration was on the whole less, but still was marked over the area of the cerebellar tracts. Somewhat lower this lateral degeneration spread into the area of the crossed pyramidal tracts, which became more markedly affected still lower down. Clarke's columns were affected, their cells scanty, their ground substance cloudy.

We shall conclude this list with two cases by DÉJÉRINE. 'Archives de Physiologie,' 1884, Nov. 15th.

(19.) Male, æt. 45. Duration of disease, six years (but 15 years before he had had some symptoms of ataxy from which he had recovered). Lightning pains. Gastric, urethral, and rectal crises. Well-marked motor inco-ordination of the legs, succeeded later in the disease by paresis of the legs, without rigidity, and with absence of tendon-reflexes. Well-marked affections of sensibility in the legs, i.e. patches of anæsthesia, analgesia, retarded transmission of sensations. Increase of the paresis to absolute paraplegia. Bedsore, pneumonia, and bronchitis, death.

Post-mortem.—Déjérine found degeneration of the cutaneous nerves in the anæsthetic parts, a condition which his researches in other cases have already brought into notice. There was *posterior sclerosis* distributed as follows: in the lumbar and dorsal regions over both the columns of Goll and the columns of Burdach; in the cervical region chiefly or solely over Goll's columns. The *lateral sclerosis* occupied in the lumbar region a triangular patch, its base at the periphery, its apex dipping into the cord, the disease being most intense at the periphery—in the dorsal region much the same area but reaching further forwards round the periphery of the cord—in the cervical region there was only a slight ring of sclerosis round the periphery.

There was well-marked meningitis, extending from the posterior columns round to the lateral, and corresponding in intensity to the intensity of the peripheral sclerosis.

(20.) Female, æt. 52. Lightning pains for several years; slight motor inco-ordination, with paresis, of the legs. Loss of equilibrium on closing eyes. Patches of anæsthesia and analgesia of legs. Exaggeration of tendon-reflexes: ankle-clonus. Death from broncho-pneumonia.

As in the last case, there was degeneration of the cutaneous nerves of the anæsthetic parts. In the cord there was *posterior sclerosis* through its whole length; but below the lumbar swelling, Burdach's columns were affected only in a very slight degree. (This gives some account of the exaggerated tendon-reflexes.) There was sclerosis of the periphery of the *lateral columns*, in

their posterior part, as far up as the cervical region. In Clarke's columns the cells were normal, but some of the fibres were diseased. There was meningitis, as in the last case.

The view taken by Déjérine of these two cases, (and he is inclined to extend it to the whole class we have been considering) is this,—they begin as ordinary tabes, i.e. systematic sclerosis of the posterior columns; in connection with this arises a meningitis, which spreads round to the lateral columns, and there sets up a sclerosis which is diffuse (i.e. limited to no particular tract), and spreads from the periphery inwards.

COMMENTARY.

These cases are susceptible of some classification, and may be subdivided according to the nature of the sclerosis in the different parts of the cord. Thus both posterior and lateral sclerosis may be systematic, or the one may be systematic, the other diffuse or of doubtful nature. (We have not included cases, in which there was sclerosis, undoubtedly diffuse, of both columns;¹ though interesting cases of the kind have been published.)

In some cases, at any rate, the sclerosis of both columns appears to have been primarily systematic, and mutually independent. Kahler and Pick's first case (No. 3) is mentioned by Déjérine as the only example of this, but he appears to have overlooked Strumpell's cases (Nos. 13, 14, 15). These, with possibly some others, (Nos. 7? 10, 17,) suffice to prove the existence, as well as the mere possibility, of multiple tract disease.

It is noticeable that in some of these (Nos. 13, 14), the lateral sclerosis was more intense than the posterior, and more strictly confined to known tracts, though usually the reverse seems to hold. The posterior sclerosis was in these two cases so symmetrical and regular as to convince the author that it could not be diffuse; yet it deviated from known tracts just so far as to suggest either that our knowledge of the developmental tracts in this region is as yet incomplete,² or that tracts of disease may differ from the tracts of development.

Usually, however, the condition appears to have been this: posterior sclerosis presumably primary and systematic, with lateral sclerosis either diffuse or of doubtful nature. We have already noticed the explanation given by Déjérine of his own cases, viz. that the sclerosis spreads from the posterior to the lateral columns, *viâ* the meninges: this explanation he would extend to several of Westphal's cases, urging, with apparent reason, that the disease of the lateral columns assumed an annular form and appeared to spread inwards from the periphery. But it appears that Westphal had already considered this hypothesis and thought it inadequate,

¹ Ballet et Minor, 'Archives de Neurologie,' No. 19, abstracted in 'BRAIN,' No. 28. Demange, 'Revue de Médecine,' Oct. 10, 1884, abstracted in 'BRAIN.'

² The development of the tracts in the posterior columns has been studied afresh by Bechterew, 'Neurologisches Centralblatt,' Jan. 15, 1885.

for such meningeal affection as there was did not correspond in position and intensity to the lateral sclerosis. He considered the latter to be primary, though not corresponding to known developmental tracts. We must be content, therefore, to say that the nature of the lateral sclerosis is in some cases doubtful. We may remark, however, with reference to peripheral sclerosis, that it may be sometimes difficult to say whether this has spread from the meninges, or is a systematic affection of the cerebellar tracts. Perhaps the integrity or the reverse of the cells of Clarke's columns may help to determine this, as these cells are held by good authorities to be the trophic centres for the cerebellar tracts.

The histology of the lesions does not appear, as yet, to have contributed much towards settling their nature. Westphal, in some of his cases, notices a difference in the histological character of the disease in the two parts of the cord: in the posterior columns there was grey degeneration, or sclerosis, characterised by overgrowth of fibrillar interstitial tissue and disappearance of nerve elements; in the lateral columns, a degeneration characterised by the presence of numerous granular cells. But these are, in his view, only different stages of the same process. Other authors, e.g. Rabesiu, mention that the minute lesions resembled those of insular sclerosis, i.e. increase of neuroglia nuclei, presence of amyloid bodies, hypertrophy of axis cylinders, &c. But D  j  rine roundly declares, that upon none of these things can we base a classification; but that the only reliable distinction between degeneration beginning in the nerve fibres and that beginning in the interstitial tissue, is to be found in their distribution; the former is systematic, the latter diffuse.

Indeed it cannot be a matter of surprise, that we know so little of the causes and of the histological beginnings of complex sclerosis, considering that our knowledge, in these respects, of the simple forms is very limited. We may begin indeed with something definite in the shape of "Wallerian degeneration," the degeneration which the fibres of a peripheral nerve undergo when separated from their nutritive centre. Comparable, if not identical with this is the process of secondary degeneration in the motor tracts of the cord when separated from their cerebral centres. But the nature of the primary degenerations is a matter of less certainty. Take the commonest of them—ordinary *tabes dorsalis*. Concerning this disease the commonly accepted view is that, since it originates and spreads in a district which is defined by nervous function rather than by anatomical characteristics, it must originate in the nerve-fibres. Yet, as readers of 'BRAIN' will remember, Dr. Buzzard has recently published a case,¹ in which, though the symptoms during life, confirmed by the appearances after death, pointed conclusively to *tabes dorsalis*, yet there was good reason to think the disease originated in the neighbourhood of the blood-vessels. This is a confirmation of the views promulgated by Adamkiewitch at the International Congress of 1881,

¹ 'BRAIN,' No. 24.

that tabes is an interstitial degeneration following the distribution of the blood-vessels in the posterior columns. But Adamkiewitch himself admits that this theory will not cover all cases, for he says in a later publication,¹ "there are two kinds of tabes, the one an interstitial degeneration originating in an overgrowth of the connective tissue which accompanies the arteries that traverse the posterior columns, the other originating from the nerves, and therefore parenchymatous in its character."²

Let us admit, however, what is usually believed, that as a rule systematic sclerosis originates in the nerve-fibres, and is therefore parenchymatous or "peritubular"; we have got to explain why particular tracts of fibres should be picked out by disease. This question is not yet removed from the field of conjecture. Shall we say that the tracts which in the course of development are latest to be completed, are the first to feel the effects of dissolution? Or shall we suppose that certain noxious agents, such, for instance, as syphilis, can centre themselves upon definite nerve districts, in the same way as the poisons of ergot and lathyrus appear to do?

With reference to the symptoms and the diagnosis, we will endeavour to summarise the hints that have been thrown out by the various authors we have quoted. Broadly speaking, it may be said that the symptoms due to lesion of either column manifest themselves so far as they are not neutralised by lesion of the other column. Assuming, for instance, the existence of a complete posterior sclerosis, we may recognise an additional lateral sclerosis (according to Westphal) by the addition of motor paresis to the symptoms of ataxia. In some such cases the paresis grows up side by side with the ataxia, modifying the ataxic gait from the beginning of the case; in others, according to Déjérine, the paralytic symptoms do not appear till quite a late period of the disease. But as to the other symptoms which usually accompany lateral sclerosis, viz. rigidity of the legs, exaggeration of tendon-reflex, ankle-clonus, &c., these, in the presence of well-marked posterior sclerosis affecting the lumbar region,³ do not make their appearance, but the tendon-reflex remains absent, as in simple posterior sclerosis.

In other cases, however, the lateral sclerosis and its symptoms predominate. This is particularly the case when the posterior sclerosis (as sometimes happens) is imperfectly developed in the lumbar region. Paraplegia with rigidity then constitutes the leading feature. If the paraplegia be complete, of course no ataxia of the lower limbs can be made out. In such cases the posterior sclerosis may manifest itself either by ataxia of the upper limbs or

¹ 'Neurologisches Centralblatt,' 1884, p. 402. Report from the International Medical Congress of Copenhagen.

² The three cases quoted from Buzzard, Ballet & Minor, and Demange, will be found highly interesting, as illustrating "peri-vascular" sclerosis. In the two first there was a history of syphilis, in the last the vascular disease was atheromatous.

³ Assuming, that is, that the posterior sclerosis involves the posterior root-zones; not merely the columns of Goll.

by sensory symptoms, such as lightning pains, anæsthesia, or the like, or by troubles of micturition, like those observed in ordinary tabes, or even by optic atrophy.

It deserves notice, that sometimes (Strumpell, No. 13 ; Raymond, No. 17) the symptoms of spastic paraplegia have been accompanied with such muscular wasting as to give the case the appearance of amyotrophic lateral sclerosis. It does not appear, however, that any reaction of degeneration has been observed.

On the whole, it seems that the elements for diagnosis of combined lateral and posterior sclerosis generally exist, and in fact the diagnosis has been more than once made.

Report.

ON RECENT ADVANCES IN THE ANATOMY OF THE NERVOUS SYSTEM.

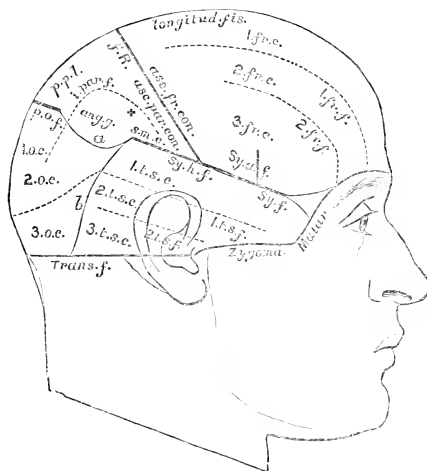
BY JAMES ANDERSON, M.D.

IN the Marshall Hall Oration of last year, after speaking of the lamentable effects of Cerebral Tumours, Dr. Ferrier asked the question: "Granting that the principles of diagnosis of reachable disease shall have become established with as near an approach to certainty as is possible, where all is hidden from the eye and hand, is there any reason why a surgeon should shrink from opening the cranial cavity who fearlessly exposes the abdominal viscera?" Just one year after it was put, the question received a practical answer in the excision by Mr. Rickman Godlee of a walnut-sized glioma from the upper part of the ascending frontal convolution. The patient was under the care of Dr. Hughes Bennett, whose diagnosis of the position of the tumour was fully confirmed by the operation. Much to the disappointment of all, the patient, after a prolonged period of steady recovery, has succumbed; but while we are thus prevented from congratulating those chiefly concerned, we hope to learn useful lessons from their account of the course of the case. Doubtless there are restrictions to cerebral surgery which do not exist in the case of abdominal surgery, but we may cherish the expectation that, with the perfecting of medical diagnosis and of surgical manipulation, will come such an advance as Dr. Ferrier in his oration ventured to predict. In order to such an advance, one of the first necessities is an accurate account of the topographical relation of the various parts of the brain to the surface of the cranium. This has been already partly supplied by the work of Broca, Heffler, Turner, and others; but the work of these observers consisted largely in laying down the relation of the cerebral convolutions to the cranial sutures, and important as that work is, it gives but little guidance either in diagnosis or operation, seeing that the sutures are but rarely perceptible through the scalp. What was wanted was the accurate laying down of the bearings of all the principal parts of the brain in relation to readily ascertained landmarks. This want has been supplied in an admirable paper published by Dr. Robert W. Reid. (*Lancet*, 27th Sept. 1884.)

The two excellent plates with which Dr. Reid illustrates his paper we are able, through his kindness, to reproduce. They largely explain themselves, and we may add, although not stated

by Dr. Reid, that they are drawn to the scale of $\frac{1}{4}$. It is to be noted as Dr. Reid says, first, that in no two brains are the sulci precisely alike; and second, that the lines and areae on the greater surface of the scalp cannot correspond accurately with the sulci and convolutions indicated by them on the smallest surface of the brain; but granting this, we may, by choosing easily felt landmarks, so lay down the bearings of any particular point of the brain that we shall be able with almost absolute certainty to include it within some part of the area exposed by a single application of the one-inch trephine. The base line (Fig. 1) is drawn through the

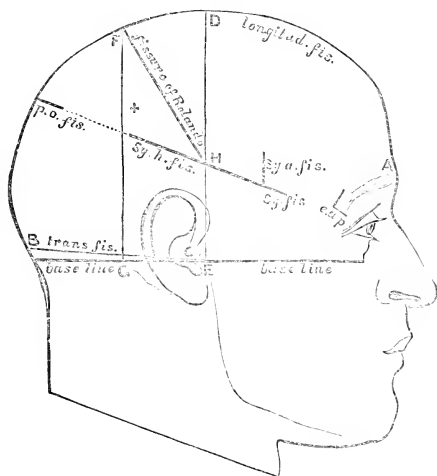
FIG. 1.



lowest part of the infra-orbital margin and the middle of the external auditory meatus, and is a definite landmark. So also are the glabella, the external occipital protuberance and the external angular process of the frontal. The most indefinite landmark used is that marked +, the most prominent part of the parietal eminence; and in many cases the posterior end of the base of the mastoid process is also very indefinite. For this reason we should prefer that the marking out of the fissure of Rolando were independent of these two points. As indicating the position of the two central convolutions it is most important that this fissure be accurately laid down, and for this purpose we prefer the method given by Dr. Arthur W. Hare in his short but most complete paper on the subject (*Journal of Anat. and Phys.* vol. xviii. Jan. 1884). Dr. Hare finds, on an average of 11 cases, that the distance from the glabella to the upper end of the fissure of Rolando is 55.7 per

cent. of the distance between the glabella and the external occipital protuberance measured along the longitudinal sinus. In no case did its variation from the point thus calculated amount to $\frac{1}{3}$ inch. Hence if the distance from the glabella to the external occipital protuberance be 11 inches, that from the glabella to the fissure of Rolando will be $6\frac{1}{10}$ inches; for $11\frac{1}{2}$ inches it will be $6\frac{2}{5}$; for 12 inches, $6\frac{3}{5}$; for $12\frac{1}{2}$ inches, 7; for 13 inches, $7\frac{1}{5}$; for $13\frac{1}{2}$ inches, $7\frac{1}{2}$. The length of the fissure, neglecting its somewhat S-shaped curvature, varies from $3\frac{1}{8}$ to $3\frac{1}{2}$ inches, and it makes on an average an angle of 60° with the mesial line of the head. These two data give us the inferior extremity of the fissure, or within less than half an inch of it. Dr. Reid first marks off the fissure of Sylvius

FIG. 2.



by drawing a line from a point of $1\frac{1}{4}$ inches behind the external angular process of the frontal, to a point $\frac{3}{4}$ inch below the most prominent part of the parietal eminence. The first three-quarters of an inch of this line represents the main fissure, the rest its horizontal limb. The ascending limb starts 2 inches behind and a little above the external angular process, and runs vertically upwards for an inch. He then deduces the positions of the fissure of Rolando and the parieto-occipital fissure by the method indicated in Fig. 1. The distance between these two fissures, according to Dr. Hare's observations, varies considerably, being on an average $1\frac{7}{8}$ inches. The method of indicating the other fissures and convolutions will be at once evident from Fig. 2. As we have said, such detailed and

accurate observations as those of Dr. Reid and Dr. Hare form the necessary foundation for any advance in cerebral surgery, and will, we doubt not, be welcomed both by surgeons and physicians.

WHEN the ordinary methods of dissection had done their best to unravel the complicated strands of the cerebral and spinal nerve-fibres, there remained yet much to be done, and to no one have we owed more in completing our knowledge than to Professor FLECHSIG, of Leipzig. His results have been obtained mainly by the use of two methods: first, that of embryology, and second, that of pathology. The first method consists in the observation of the different groups of fibres which acquire their medullary sheaths at the same period; the second is the marking out of tracts of fibres by their secondary (Wallerian) degeneration centrally or peripherally after section or injury, and also in some few cases by the non-development of fibres owing to the congenital absence of their centres. These results Professor Flechsig has summarised in his 'Plan of the Human Brain' (*Plan des menschlichen Gehirns*; Leipzig: Veit & Co., 1883) with its accompanying explanatory text. It will not be expected that we should criticise this plan in detail. Indeed great part of it is now beyond criticism, being incorporated among the well-ascertained facts of neurology. His account of the crusta and tegmentum of the crus cerebri is now substantially accepted, while there may still be doubt as to the exclusively cerebellar connection of the inner and outer portions of the crusta. He has also doubtless, as Professor Wernicke in a recent criticism remarks, attributed to the frontal and temporo-occipital zones a too purely mental function as opposed to the sensori-motor parietal lobe. He lays very great weight, from a physiological point of view, upon the relation of the sutures of the skull to the underlying cerebrum. This can hardly be said to be substantiated.

Alongside of Flechsig's 'Plan,' we have Professor Aebly's Scheme of the Nerve Tracts in the Human Brain and Spinal Cord' (*Schema des Faserverlaufes von menschlichen Gehirn und Rückenmark*; Bern, J. Dalp, 1884) which has already reached its second edition. It agrees in all large facts with that of Professor Flechsig, but is in many respects more complete than his and more easily intelligible. It gives both a transverse and antero-posterior section, besides subsidiary sections of the crus cerebri and the fourth ventricle. The work is accompanied by an admirable explanatory text, which closely agrees with the account of the central nervous system given by Professor Wernicke in his work on Nervous Diseases, a detailed criticism on the anatomical part of which, by Dr. James Ross, will be found in a previous volume (BRAIN, Oct. 1883, p. 398).

PROFESSOR BURT WILDER in his recent Cartwright Lectures at New York (*New York Medical Journal*, Feb. 9th, 1884, *et seq.*) tells us how to preserve, examine and describe the human brain. This is a subject on which we need instruction (sorely need it, in fact), for Dr. Wilder's amusing account in his second lecture of what happens

in the dissecting-room on the removal of the calvarium will doubtless recall familiar if not pleasant memories to those who read it. Our modes of preserving and studying the brain are undoubtedly crude and unsatisfactory, and Dr. Wilder's lectures, which we have read through with considerable care, contain many points which indicate that could we but spend a day with him in his laboratory, we should learn from him much that would prove practically useful. Before deriving benefit or information, however, from Dr. Wilder's lectures, the reader will find that he has a new nomenclature, in fact almost a new language, to learn. In removing the brain he will find that he is directed to make "a hemisection a little laterad of the meson," the meaning of which he may guess at, but will not be at all certain of. Again, he will scarcely recognise the familiar third ventricle as the *diacele*, or the cerebellum as the *epen*; and to be told that a new and little-known structure lies "dorso-cephalad" of the *calcar*, may not, without explanation, convey a definite idea of locality to his mind. As far as the actual development and comparative anatomy of the brain is concerned, we must frankly say that the reader would hardly be repaid by Dr. Wilder's lectures for the trouble of mastering his nomenclature, as he will find a more compact and intelligible account of it in Huxley's 'Anatomy of Vertebrates,' to which, along with the list of names in Quain's 'Anatomy,' Dr. Wilder acknowledges his indebtedness. Having said this, however, we can cordially recommend a perusal of Dr. Wilder's lectures, embodying as they do the detailed, persevering, and most loving study of an organ, which well repays such study even from a more or less strictly morphological view-point. He recommends that the brains of various animals should be used for practice,—those of the frog, necturus and cat, with embryonic brains, being principally used in his laboratory. For practice, it may not be so necessary in this country to follow his advice; but just as every student of medicine should more or less thoroughly dissect two or more animals besides man, in order fully to appreciate human anatomy, so specially he ought to dissect the brains of several animals. The human brain is, as regards the general animal kingdom, almost a monstrosity, owing to the huge development of the cerebral hemispheres, and its true morphology can be learnt only from lower and less complicated brains. The removal of the brain intact from the cranium is an operation requiring some skill and more than some practice. We feel tolerably certain that Dr. Wilder's method of supporting the brain in brine would in most hands result in disaster. The continuous injection with alcohol (continuous "al injection") of the brain *in situ* has much to commend it, if the bleaching of the grey matter, which is its main disadvantage, be corrected by subsequent observation of the fresh brain. The importance attached to the description of the cerebral cavities by Dr. Wilder is a point in which he differs from our ordinary works on the anatomy of the brain, and his method of injecting the ventricular cavity from the infundibulum is devised in order to obtain a more exact impression of the shape and relation of these

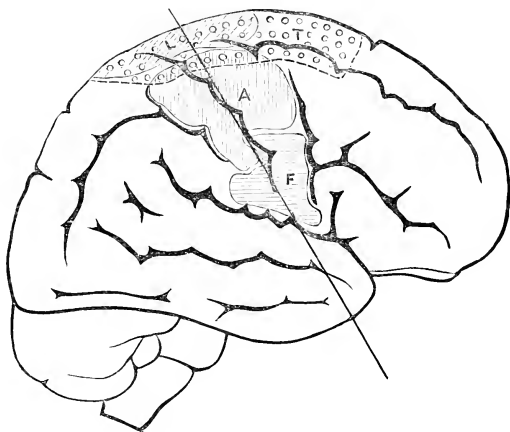
cavities. In Dr. Wilder's third and most important lecture, which we shall notice in a subsequent issue, he proposes to discuss the prevailing errors and omissions in the ordinary descriptions of the brain, along with points in cerebral anatomy still requiring elucidation.

OUR knowledge of the functions of the cerebellum may be roughly summed up, by saying that it is in some way concerned in the co-ordination of movements. How it is concerned in this, and whether it has any other function, we do not know with any certainty, and neither physiological experiment, nor pathological lesions, have in recent years much advanced our knowledge. Professor SPITZKA, of New York, has recently (*Alienist and Neurologist*, Jan. 1884, p. 92), by a happy reversion to a method from which we had ceased to hope for further light, attempted to solve the problem by a study of its comparative anatomy and its anatomical relations. He points out that the cerebellum steadily increases in size and complicacy as we rise in the scale of animals, becoming gradually more and more overlapped, however, by the backward-projecting cerebrum. This marked and concomitant growth of the cerebellum is a cardinal point, which we must not allow to be concealed from us by the complete overlapping of the cerebrum in the anthropoid apes and man. The vermiiform process alone is represented in reptiles and birds, and in most mammals this forms a markedly separate central lobe, with its own grey nucleus, the *nucleus fastigii* in the roof of the fourth ventricle. In man this central lobe is dwarfed by the great development of the cerebellar hemispheres, with its complicated grey cortex and its grey *corpus dentatum* in the centre. The fibres connected with the cerebellum are, (1) The three crura which Professor Spitzka prefers to term tegmentibrachium, pontibrachium, and myelobrachium; (2) the trapezium, or deep transverse fibres of the pons; and (3); the auditory nerve. Of the function of the trapezium nothing definite is known; but otherwise he points out that the cerebellar connections are afferent from periphery to cerebellum, or efferent from cerebellum, or efferent from cerebellum to cerebrum; and he considers that a direct motor innervation can be entirely excluded from the functional possibilities of the organ." Through the restiform column it is connected, Professor Spitzka holds, with the posterior column of the cord, decussating by means of the olivary nucleus, and with the centripetal direct cerebellar tract. On the inner side of the restiform column is a tract which he supposes to come direct without decussation from the postero-external column of the cord. A bundle from the pneumogastric nerve has been traced to the flocculus, and Professor Spitzka has confirmed the existence of a direct cerebellar root of the sensory division of the trigeminus. The connection of the auditory nerve with the cerebellum he considers as most important in discussing the function of the cerebellum. In the lower animals, even with a rudimentary cerebellum, this auditory connection is extensive, and "with higher development the general sensory periphery and

auditory apparatus attain a more extensive projection; and this projection grows step by step with the cerebrum itself." Meynert points out that the cerebral hemispheres, the cerebellar hemispheres, the dentated nuclei of the cerebellum, and the olivary nuclei hypertrophy in proportionate degree as we proceed upward. Summing up his conclusions, Professor Spitzka says: "In other words, the cerebellum is a field where impressions of touch and position are associated with those of time and space. *Per se* this organ can do nothing, it is merely an informing depôt to the great head centre, the cerebrum. Whatever molecular oscillations are determined within its ganglionic substances must pass to higher centres for translation to skilled motor reaction."

Abstracts of British and Foreign Journals.

On Substitution. By VICTOR HORSLEY, B.S., F.R.C.S. (*Lancet*, July 5th, 1884).—After discussing the theoretical question of substitution or compensation, Mr. Horsley refers to the results of an experimental research conducted by himself and Professor



Right hemisphere of the cerebrum, showing the centres in the excito-motor area of the cortex arranged round the fissure of Rolando in the order described in the text. F. faeial centre. A. Centre for upper limb. L. Centre for lower limb. T. Centre for trunk muscles. This centre being really on the mesial surface of the hemisphere, and so in the longitudinal fissure, is represented at its border by a dotted line. The straight line represents almost the plane of section shown in Fig. 2, page 81.—Cf. Fig. page 100.

Schäfer (*Proc. Royal Society*, 1884) on the subject of cerebral localisation. These observers have arrived at certain conclusions, which are modifications of previously received doctrines, as well as advanced a step in some particulars. For example, they place

the cortical centre of the lower limb at the upper extremity of the fissure of Rolando, extending backwards into the parietal lobule and forwards into the superior frontal gyrus, which is a somewhat different position from that in which it is assumed to exist by Ferrier. The list of cortical areas is completed by finding that the muscles of the trunk are regulated by centres which lie in the marginal convolution on the mesial surface of the hemisphere. They have shown that the centre for the lower limb dips largely over into the marginal convolution opposite to the upper end of the fissure of Rolando. Below these centres in order, as was before determined, come those for the upper extremity and face, and it is maintained that the fibres in the direct pyramidal tract preserve the same relative position.¹ "When opposite the centre of the lenticular nucleus the fibres become bent in entering the area of the internal capsule, and being twisted like the rays of a fan close to its hinge, now become arranged in an antero-posterior manner, the most anterior being what was before the most inferior." A hæmorrhage of the lenticulo-striate artery, which courses round the under surface of the lenticular nucleus passing through it and the internal capsule to the caudate nucleus, will destroy the pyramidal fibres in the order of face, upper limb, lower limb, and trunk, as the focus of disease will be below and in front of the bundles of fibres. This is exactly the order which is observed to be affected clinically. This arrangement of fibres from the cortical centres in the internal capsule is strikingly exemplified by a case in this number of 'BRAIN,' in which a flat limited softening in this tract caused complete paralysis of the upper extremity without any involvement of the face or leg.

Further it will be seen that Mr. Horsley's explanation accounts for the classical mode of recovery of power in hemiplegia, without resorting to the well-known theory of substitution advanced by Dr. Broadbent; the two factors being: 1. Removal of pressure by absorption of extravasated blood, &c.; 2. Further development of function in the bilaterally acting cortical centres.

Sharkey and Lawford on Acute Optic Neuritis, associated with Acute Myelitis. (*Ophthalmological Society's Transactions*, vol. iv.)—A girl, aged 17, previously in good health, rapidly lost her sight, so that in four days she was quite blind, without other prominent symptoms. Well-marked double optic neuritis was

¹ See Figures on pages 81 and 82 of the present number of 'BRAIN.'

found to be present a month afterwards. Symptoms of paralysis and loss of sensation in the lower extremities supervened. About three weeks afterwards the patient died from symptoms of peritonitis; that is sixty-two days from the time vision first failed, and twenty-nine days after the first appearance of symptoms of paralysis. A very careful account of the post-mortem examination is given in detail, showing the brain to be healthy. The spinal cord presented the appearance of acute myelitis at the lower cervical and upper lumbar regions. The optic tracts and nerves, along with the discs and retinæ, were accompanied by all the evidences of intense inflammatory change.

It is pointed out that the interest in this case lies in the association of an acute optic neuritis with acute inflammation of the spinal cord. There is probably some relation between the two, although the one does not depend directly on the other. In this case the optic neuritis appeared a month before the spinal symptoms. Post-mortem examination showed that the spinal cord between the lower cervical and lumbar regions was healthy, so that the centres of disease must have originated independently one of the other. The same may be probably asserted with regard to the optic nerves. Other cases have been published, by Clifford, Allbut, Seguin, Noyes, Steffen, and Erb, in which spinal symptoms were associated with changes in the optic discs. In the *Archives of Ophthalmology*, for 1882, No. II., a case is recorded by Julian J. Chisholm, M.D., somewhat similar to the one under consideration, but without post-mortem examination. In the *Lancet* for 1882, Dr. Dreschfeld published two cases, each with an autopsy, of acute cord disease, accompanied with optic neuritis.

These cases moreover prove, that the occurrence of double optic neuritis in association with nervous disturbance is not of necessity due to cerebral disease.

Sharkey on Embolism of the right middle Cerebral Artery, producing left Hemiplegia and Hemianæsthesia, Absorption of a large portion of the Right Hemisphere: Death seven years later. (*Transactions of Medico-Chirurgical Society*, vol. lxvii. 1884.)—A woman, aged 34, with heart disease, was seven years ago seized with an apoplectic attack, followed by left hemiplegia and hemianæsthesia, including amblyopia of the left eye and deafness of the left ear. She gradually improved, and in about four months, although the arm remained almost useless, very fair power had returned in the leg, and sight and hearing were completely

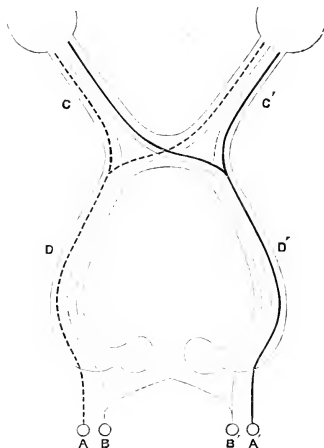
restored. Seven years afterwards she again came under observation. During this interval she had been somewhat weak-minded, but remained, according to her husband, a good-tempered, hard-working, and affectionate wife. On examination, there was weakness and rigidity of the left upper extremity, and paresis of the left leg and side of the face. The sensibility was everywhere normal; hearing and sight were as in health. The patient died suddenly. After death, in addition to heart, liver, and kidney disease, the brain was found extensively altered. The morbid condition may be summed up by saying that in the right hemisphere there was great deficiency in its substance. The convolutions affected were those supplied by the Sylvian artery, viz. the inferior frontal, the external portion of the orbital, the lower half of the two central convolutions, the inferior parietal lobule, the convolutions of the island of Reil, the angular gyrus, the inferior and middle temporo-sphenoidal, and part of the inferior occipito-temporal convolutions. These convolutions had completely disappeared, and a depressed, ill-defined, loose mass of a yellow colour remained. The right optic thalamus, lenticular nucleus, both corpora quadrigemina, and the right half of the pons, and right pyramid, were reduced in size. Drawings are given of the anatomical appearances.

The author points out that this case is one of embolism of the right middle central artery, occurring seven years before death, and resulting in subsequent absorption of those parts of the brain which are supplied by it. The chief features of interest are, that although so large a portion of one cerebral hemisphere was absent, the patient remained a useful member of society for seven years. Again, although at first there was total hemiplegia and hemianæsthesia, the loss of motion alone was permanent. Owing to the great destruction of the basal ganglia, the case is useless for purposes of cortical localisation. Still some important conclusions may be drawn. 1. Destruction of the angular gyrus and superior temporo-sphenoidal convolutions, on one side, does not involve permanent loss of sight and hearing on the other. 2. Each hemisphere is specially connected with the sight of the opposite eye. 3. Sensation on each side of the body is not so indissolubly connected with certain definite areas of the opposite hemisphere as is the case with motion.

Sharkey on Homonymous Hemianopia, probably due to a Cortical Lesion. DR. SHARKEY (*Ophthalmological Society's Transac-*

tions, vol. iv.) relates the case of a woman, aged 51, who had suffered from paroxysmal seizures for two-and-a-half years. These consisted of a play of colours in the right eye, followed by convulsion of the right hand and arm, subsequent rigidity of the leg on the same side, and finally loss of consciousness. After the original attack there was paresis of the right arm, and inability to see on the right side, without optic neuritis or other special nervous symptoms. Details of the case are given, and among others the fact, that the defect of vision did not reach as far as the vertical line drawn through the fixation point, but occupied only the peripheral field of vision on the same side in both eyes. There was no post-mortem examination, but the conclusion drawn from all the circumstances of the case was that the lesion existed in the left hemisphere, affecting the cortical centre of the arm and its neighbourhood. Immediately posterior to these are the angular gyrus and occipital lobe, both which are supposed to contain the central elements of the sense of sight. Hence the same disease might easily involve both these motor and sensory areas. Both physiological experiment and pathological observation point decisively to the view, that each hemisphere is connected with both eyes, and that there is to some extent a separation between the area in the cerebral cortex which receives the peripheral fibres of the corresponding halves of the retinae, and the area which receives the centre fibres of the opposite retina. The course which link the fibres of the retina to the cerebral cortex have been best demonstrated by an appeal to pathological observations. It has been for some time known that diseases of the optic nerve caused complete blindness of the same eye, and that destruction of the optic tract caused homonymous hemianopia, in which the dividing line passes through the fixation point. The case under notice, however, shows that disease of some portion of the cerebral cortex produces homonymous hemianopia confined to the peripheral portions of the retinae, their central parts retaining clear vision. Hence the fibres of the optic tracts as they pass into the hemispheres must separate in some such way that those which come from the periphery of the retina enter the cortex at a point which is, at least to some extent, distinct from that which receives the central fibres. Pathological and experimental observations moreover prove, that each hemisphere has an essential and well-marked special connection with the opposite eye; and as separate areas in both hemispheres supply the peripheral fibres of each eye, this special

connection can only take place by means of the central fibres. Now lesions of the optic tracts prove, that the central fibres of each retina are contained partly in the tract of the corresponding side, partly in that of the opposite side. Therefore those in the tract of the same side must cross beyond the corpora geniculata, in order to reach the cortex of the hemisphere opposite to the eye



from which they come. The accompanying diagram, which is a modification of Charcot's, is given to graphically represent this reasoning. The case under consideration supports the view, that in homonymous hemianopia in which central vision is retained for some degree on all sides of the point of fixation, the cause is probably cortical.

A. HUGHES BENNETT.

Disseminated Sclerosis and Infectious Diseases. (P. MARIE, *Progr. Médical*, Apr. 12, 1884, *et seq.*)—M. Marie opens his paper with the following case. Two years after typhoid fever, certain nervous symptoms made their appearance; these were, slow and hesitating speech, not scanned. Tremors, nystagmus, homonymous diplopia. Staggering gait. Some paresis of limbs. Increased knee-jerk, no loss of sensation. He regards these nervous symptoms as indicating the pathological condition known as dis-

seminated sclerosis, and he considers that the typhoid fever is the cause of them.

In support of this view, he quotes a considerable number of cases recorded by competent observers. Of these, 10 cases are given in which the nervous symptoms appear after typhoid fever; 4 after variola; 2 after erysipelas; 3 after pneumonia. He also gives cases occurring after measles, scarlatina, whooping-cough, intermittent fever, diphtheria, cholera, syphilis. In all these cases the prominent symptoms were tremor, paresis of limbs, and, less often, troubles of speech and nystagmus.

In only three cases (Ebstein, Jolly, Joffroy) was the diagnosis confirmed by autopsy.

In five cases the symptoms abated, or completely disappeared.

The author ends with some remarks on the pathology of disseminated sclerosis. He starts by postulating the vascular nature of the disease, and he remarks that arteritis bears a close relation to infectious diseases, and also tends to be disseminated over the body in patches. Popoff has described an accumulation of lymph-cells in the perivascular lymphatics in typhoid fever. Ribbert believes that an irritant substance (microbe) can be carried by the circulation, and being arrested by the vascular wall, determines a perivascular inflammation, which is the cause of a patch of sclerosis.

M. Marie arrives at the conclusion, that typhoid fever and other infectious diseases have a series of secondary and tertiary accidents, comparable to those of syphilis. As one of these he regards disseminated sclerosis. He suggests that what are now called "*late complications*" of the diseases, should be called more properly "*late manifestations*."

M. Marie's paper is a very interesting one, but we must admit that, however much we may be inclined to agree with his conclusions, yet we cannot regard his premisses as particularly strong. It is much to be regretted that more autopsies are not recorded. We should decidedly hesitate to diagnose disseminated sclerosis from the meagre symptoms in some of the quoted cases.

However we cannot but hail with interest any observations on the aetiology of so obscure a disease as disseminated sclerosis, and M. Marie's remarks on the pathology of the disease are particularly instructive.

H. H. TOOTH.

Case of Recurrent Dropsy of the left middle Ear, complicated after eight years duration by acute attack of Monocular Optic Neuritis on the same side, followed by General Tabetic

Symptoms. By CHARLES H. BURNETT, M.D., and CHARLES A. OLIVER, M.D. (*American Journal of Medical Sciences*, Jan. 1884).—At 8 years of age the patient had a polypus removed from the right ear; when seen at the age of 55 he was deaf with this ear, and it appeared that the canal had become closed by the growth and organisation of granulation tissue formed after the removal of the polypi. He applied, however, on account of recent deafness in the other (left) ear. The membrane was opaque and the malleus retracted. But from statements of the patient it was surmised that there was fluid in the tympanic cavity: paracentesis evacuated a little tea-coloured fluid, and gave much relief to the symptoms.

Deafness, however, recurred, and the operation was repeated with relief to this symptom as many as 38 times in the course of the next two years. At last the fluid did not return, but ordinary hypertrophic catarrh of the middle ear set in. He had by this time developed dizziness, and staggered on turning round. This symptom, however, the authors regard as “tabetic in origin” rather than as referable to the ear-disease.

About a year after the aural treatment was begun, he complained of dimness of vision in the left eye. In this (the left eye) there was well-marked optic neuritis with swelling of the disc. Vision for light only. In the right eye the optic disc was dirty grey in colour, but there was no evidence in the authors’ opinion of past neuritis: there was uniform contraction of the colour fields. The optic neuritis of the left eye gradually disappeared; the disc finally assuming the dirty-grey colour seen in the right disc, and correspondingly the vision for form and colour reappeared, to disappear again as atrophy set in. In brief, simple atrophy was observed in the right optic nerve, optic neuritis with consecutive atrophy in the left.

The remaining symptoms observed were, loss of smell in the left nostril; unsteadiness when walking, exaggeration of patellar tendon-reflexes, especially on the left side; which subsequently became diminished, though the left remained always in excess of the right. The pupils acted normally to light. There was slight paresis in the right facial district, probably to be connected with the old ear-disease on that side.

The condition of the ears the authors suppose to be purely local. The uniocular neuritis and the loss of smell they ascribe either to a cerebral tumour of uncertain position or preferably to a chronic pachymeningitis at the base of the left side of the brain; the remaining symptoms to sclerosis of the posterior columns. (But

seeing that the tendon-reflexes were exaggerated, the pupils acted normally to light, and that there were no pains, the diagnosis of posterior sclerosis seems rather uncertain.)

On the neglect of Ear-symptoms in the Diagnosis of Diseases of the Nervous System. By G. L. WALTON, M.D. (*Journal of Nervous and Mental Disease*, October 1883).—The author draws attention to the fact, that while the relations of the eye and its functions to nervous disease have been minutely studied, and the condition of this sensory organ is reported in all careful accounts of cerebral disease, yet the ear and the sense of hearing have been for the most part neglected.

Hysterical deafness, with loss of hearing power for high tones, analogous to hysterical blindness with contraction of the visual field and colour-blindness, is, he thinks, an example that ophthalmic facts, important from a neurological point of view, may be paralleled by aural facts. But, as a matter of fact, in the reports of nervous cases the hearing power is usually either not inquired into at all, or else any existing deafness is at once set down to the nervous disease without investigation of the peripheral organs of hearing. Thus, the seeming rarity of deafness in disease of the pons and cerebellum is probably due to the fact that the hearing is not systematically examined in such cases. Actual examination is necessary, for the patient may be quite ignorant that he is deaf, and deafness of one ear may evade the physician's notice if not looked for. But even where deafness is reported, there is often either no examination of the ears made, or a most superficial one. Thus, cases of locomotor ataxy with deafness are frequently related; the inference being that the deafness is due to the nervous disease; whereas, according to the author's experience, it is much more often peripheral than central. The same with the so-called Ménière's disease, which is more often than not due to disease of the middle ear.

Examination of the Spinal Cord in a Case of Polio-Myelitis of the Adult of two months standing. By JAMES J. PUTMAN, M.D. (*Journal of Nervous and Mental Disease*, January 1883).—A single woman, æt. 22, had caught cold while menstruating, three weeks before admission; the flow was checked, and pain in the head, neck, back, and limbs set in, with vomiting. In three days there appeared paralysis of the limbs, with numbness, and inability to hold her urine.

When first examined, the temperature was 99.8° ; there was tenderness along the spine, pain on movement, atrophy of the muscles of the limbs, specially marked in the right arm and intrinsic muscles of the right hand; some contracture of the right arm and hand. There was some improvement for a month; then a rise of temperature, up to 102° . After another month there came a similar rise of temperature, accompanied by vomiting and diarrhoea, which proved fatal.

The post-mortem appearances (naked eye and microscopic) are thus summarised by the author. Extensive ulcerations in the large intestine; subpleural hæmorrhages. Throughout the whole length of the spinal cord anterior and posterior poliomyelitis [each anterior and posterior cornu was threaded in its whole length by a column of inflamed tissue]; but the right anterior cornu had suffered most severely; here, in many sections, there were no healthy ganglion cells to be seen; also atrophy of the anterior nerve-roots and to a less extent of the posterior; subacute inflammation of the antero-lateral white columns; a moderate amount of lepto-meningitis; thickening of the vessels everywhere even in the posterior columns, and diffuse though moderate increase of the connective tissue.

The author suggests that diffuse myelitis would be the fittest name to apply to the case.

The Pathological Anatomy of the Cerebro-Spinal Axis of a Case of Chronic Myelitis of nineteen years standing. By Dr. H. D. SCHMIDT (*Journal of Nervous and Mental Disease*, July 1883).—The case originated in a gunshot-wound of the neck. At the time of the post-mortem there was no trace left of fracture of the vertebræ, or of direct injury to the cord in this situation. Complete paralysis of all four extremities succeeded the injury immediately. The paralysis of the legs disappeared, leaving the arms paralysed, and at first hyperæsthetic, subsequently anæsthetic. The arms became contracted in a year. Sixteen years after the injury the patient could walk, but there was contracture of the left foot, and commencing pains and hyperæsthesia of the lower limbs. After 18 years, muscular atrophy had begun in the hands and arms. He finally became unable to walk, lost control over his evacuations, and died in a state of coma and complete paralysis.

The macroscopic appearance of the brain was normal. The cord in the cervical region was flattened and softened, there was here a tubular cavity apparently caused by the softening.

Further, a yellowish-grey discoloration was observed in the posterior columns, in the left postero-lateral column and in the periphery of the white substance, and in spots scattered through the rest of the white substance. It diminished from above downwards, and does not appear to have been strictly systematised; it was generally diffused through the pons and medulla. Microscopically, the following points were made out. (*a*) There was degeneration of almost all the vessels of the cord—the arteries of the pia mater (which were least affected) showed thickening of the adventitia and indistinctness of the muscular coat, while the walls of the smaller vessels within the cord and medulla were pale, granular and indistinct. (*β*) Round the vessels, both upon the large and small septa of the cord, was a fibrinous exudate, pale and granular, but staining deeply with carmine. (*γ*) There was atrophy of nerve fibres very variable in degree, apparently caused by pressure from the exuded material. (*δ*) Numerous rounded bodies were observed, both in the grey and white matter, specially in the medulla and pons. These could not be proved chemically to consist either of fat or of amyloid substance. The author considers them to have been ganglion cells of the grey matter and nerve-nuclei of the white substance in various degrees of degeneration.

Two Cases of compression of the Spinal Cord by Sarcomatous growths from the Soft Membranes. By G. LONG FOX, M.D., F.R.C.P. (*Bristol Medico-Chirurgical Journal*, July, 1883).—*Case 1.*—A woman æt. 35, strained herself while lifting a window, and a month after began to feel pain in the right arm and weakness in the right hand. Some months later, after getting wet, loss of power in both legs and arms was noticed. When seen nine or ten months after the original strain, there was almost complete motor paralysis of both arms, sensation being also impaired, though not absent; and complete motor paralysis, with dulness of sensation, in the legs. There had been incontinence of urine and constipation. Pupils regular. Tendon-reactions exaggerated. No distinct muscular atrophy. Some tenderness over 6th cervical vertebra (a seton had been worn there). Death in five months more, from bronchitis and paralysis of the intercostals.

Post-mortem, a hard rather vascular tumour (having the microscopic characters of a spindle-celled sarcoma), measuring $1\frac{1}{2} \times \frac{3}{4}$ in. was found growing from the arachnoid on the anterior surface of

the cord in the cervical region. It had there destroyed by pressure all the cord except the posterior columns. The cord was almost deliquescent at the level of the tumour, and softened down to the lumbar region.

Case 2.—Shooting-pains in the arms, especially at night, for three months, the right arm being worst: then loss of power of extension, first in the right fingers, and next in the left. For five weeks, numbness and pricking in the toes, first of the right foot, and then of the left; gradually this extended upwards to the waist. Priapism and difficulty in micturition. When examined, there was anaesthesia up to the third rib, and paralysis of the lower limbs. Plantar reflexes normal. The fingers were at first the only parts of the upper limbs that were paralysed, but gradually nearly the whole arms became involved. Thoracic respiratory movements began to fail before death.

Post-mortem.—Well-defined tumour (spindle-celled sarcoma), measuring $1\frac{1}{2}$ inch \times $\frac{3}{4}$ inch, on the left posterior aspect of cord in cervical region. Much softening of the cord in its neighbourhood. Along the whole length of the cord below was a belt of yellowish-grey substance $\frac{1}{4}$ inch thick, enveloping the cord on all sides equally.

Seguin on the American method of giving Potassium Iodide in very large doses. (*Arch. of Medicine*, 1884, p. 114.)—The author alludes more particularly to the later nervous lesions of syphilis. In many cases he admits that ordinary doses (*viz.* up to 8 grammes, or 120 grains daily) are sufficient. But it often happens that 10 or 15 grammes a day have to be administered, and this dose increased week by week, before results can be obtained. For instance, in syphilitic headache Seguin prescribes two doses of 4 grammes (60 grains) each on the first day, and increases this quantity by one dose every day until 32 grammes (1 ounce) are taken in the twenty-four hours. Very large quantities must be taken in syphilitic coma, especially when convulsions or choked-disc are present. It is obvious that in specific hemiplegia, and the like, the iodide cannot be expected to remove the symptoms depending upon actual destruction of nervous tissue. The salt should always be given on an empty stomach, largely diluted with some alkaline water.

On the efficacy of Iodide of Potassium in non-Syphilitic Organic Diseases of the Nervous System. By Dr. E. C. SEGUIN.—The author gives three classes of cases.

I. In which symptoms were temporarily relieved, and in which, post-mortem, the lesion was proved to be non-syphilitic.

Case 1.—Right-sided hemiparesis with inco-ordination, palsy of left external rectus; headache, vomiting, optic neuritis. Improvement under iodide of potassium (10 to 40 drops of a saturated solution three times daily for two months). Death about five years afterwards: *sarcoma of left crus cerebri*.

Case 2.—Headache, vomiting, staggering gait; exophthalmos, double optic neuritis; a soft pulsating tumour in region of lambdoid suture. Iodide from 90 to 150 grains per diem; some relief to symptoms. Death within a few months: *jibro-sarcoma of cerebellum, with dropsy of the ventricles*.

Case 3.—Fall on back of head; subsequently attacks of vomiting with headache: paralysis, especially of the left side, optic atrophy. Four years later, right hemiparesis, absence of tendon-reflex at knees, epileptiform attacks with occipital pain. Iodide (10 to 40 drops of a saturated solution), taken three times daily, cut short the fits. Death next year. *Sarcoma of right hemisphere of cerebellum; meningitis of convexity of cerebrum*.

II. Cases without post-mortem; symptoms cured or relieved by iodide, no evidence of syphilis.

Case 1.—Numbness of left side, mental affection. Numbness disappeared while taking iodide; patient lapsed into dementia paralytica.

Case 2.—Paralysis of third nerves; paresis and ataxia of limbs. Two attacks were relieved by iodide of potassium, 30 to 120 drops of the saturated solution three times daily. A third attack appears to have resisted all treatment.

Case 3.—Right-sided convulsions affecting the face principally; aphasia. Recovery while taking iodide of potassium, at first combined with the bromide.

III. Cases of double optic neuritis in children, probably due to basal meningitis; apparent good results from large doses of the iodide. Three cases are given; headache, vomiting, and internal squint were observed in addition to the optic neuritis.

The author gives the iodide largely diluted with water, Vichy water, or solution of bicarbonate of soda.

J. A. ORMEROD, M.D.

B R A I N .

JULY, 1885.

Original Articles.

ON THE CORPUS CALLOSUM IN THE EMBRYO.

BY D. J. HAMILTON, M.B., F.R.S.E.,

Professor of Pathological Anatomy, University of Aberdeen.

IN a paper which I communicated to the Royal Society on February 23, 1884, I detailed certain facts and figured certain appearances in the *adult* brain which led me to believe that the Corpus Callosum is not an interhemispherical commissure, as is generally supposed, but in reality the decussation of a great part of those fibres derived from the cortex which do not decussate at some point further down. The facts were mainly these, that when the brain is prepared in the method I employ (see 'BRAIN,' July 1883) the fibres of the corpus callosum, instead of stretching across from side to side between the hemispheres, are found to come from the cortex of one side to pass over to the opposite side, and having gained this, to turn down into the inner and outer capsules. I endeavoured to show that the bulk of the fibres which enter the inner capsule, both in its anterior and posterior segments, is composed of such crossed callosal bundles, while the motor and other fibres which come chiefly from the cortex of the same side, which do not pass over in the corpus callosum, and which decussate further down, constitute but a small part of the entire inner capsule.

The outer capsule, I demonstrated, is composed of two layers—an external and an internal. The external derives its fibres

from the operculum and edge of the Sylvian fossa behind this, while the inner is made up mainly, if not entirely, of crossed callosal fibres, which have come from the opposite side, and turned downwards.

As regards the destination of these crossed callosal fibres which have thus passed downwards into the two capsules, I pointed out that they chiefly terminate in the thalamus opticus. A few of them end in the caudate nucleus, while a considerable number, in all probability, find their way further downwards to end in the pons Varolii or other masses of grey matter below the basal ganglia. The majority, if not all the motor fibres do not pass through the corpus callosum; they belong to a different system, and, partly, at least, are continued down through the pyramids to the spinal cord as usually described.

There are therefore two main sets of fibres entering the inner capsule from the cerebral cortex; the one set comes from the opposite hemisphere, decussates in the corpus callosum, and subsequently enters the capsule: the other set, much the smaller of the two, passes into it from the cortex of the same side, and decussates somewhere lower down, chiefly in the medulla oblongata.

The facts supporting these views were mainly derived from the analysis of the nerve tracts in the *adult* human brain, and since then I have confirmed them, over and over again, in numerous observations. I find, moreover, the same appearances in all animals which I have examined, in which an undoubted corpus callosum is present. So far as I have gone, these comprise the ape, monkey, horse, sheep, dog, cat, and pig.

For the purpose of confirming or refuting the foregoing views, several methods of inquiry naturally suggest themselves, and among those which I have employed, and am at present employing, are the study of the corpus callosum in the embryo, the examination of it in destructive lesions in the human cortex, and experimental research in the lower animals. It is with the first of these—the corpus callosum in the embryo—that the present communication is chiefly concerned. The brains which I have employed in this present research, so far as it has gone, have been derived from the embryos of various

animals, and from the human foetus. The latter, as is well known, has yielded in the hands of Flechsig the most brilliant results; and although my observations in some respects lie in a different path from those of that observer, yet I have also found that the human foetal brain is excellently well suited for such investigations. The great difficulty, however, lies in getting the materials sufficiently fresh, for it will be found by those working at the subject, that unless perfectly fresh, the embryonic brain-substance loses that consistence which is necessary to hold its parts together. The embryos of mammals are also of course extremely useful in such an investigation, and as the majority of those slaughtered for food purposes can be obtained in various stages of foetal life, I have employed these largely in conducting the present inquiry. I have examined the foetal human brain at almost all ages, but that age which I would specially recommend as being most suited to demonstrate the connections of the corpus callosum, is *from the end of the third to the fourth month*. It is difficult to say, of course, in the human subject what the exact age of a foetus may be, and hence Flechsig has fallen back upon length as a surer basis to reckon by. Foetuses, however, differ so much in length at the same period of intra-uterine existence, that even this is to a certain extent misleading, and hence perhaps the various discrepancies in Flechsig's account of the period of medullation of the various nerve-fibre tracts.

The chief advantage of studying the corpus callosum in the embryo, I have found, lies in the fact, that its fibres are developed long before those which enter the inner capsule directly from the cortex of the same side. Hence those fibres which are callosal can be clearly traced in the embryo, apart from others with which they become associated and intermingled in the adult. It is, however, only at a certain period, the above mentioned, that this can be satisfactorily accomplished. Previous to this, the fibres are too rudimentary to afford good results, and, afterwards, the direct cortical tracts and the "association system" of fibres become so highly developed that the callosal bundles are lost, or are indistinctly demarcated from them. In the adult brain I know of no appearance in the nerve centres so fallacious as that of the

course of the callosal fibres after they have crossed. Looking at a perpendicular section of a fresh brain, or one hardened in spirit, it is absolutely impossible to say definitely where they go to; and hence the idea has gained almost universal credence, that they pass from the cortex of one side to that of the other, simply because the corpus callosum lies between the two hemispheres. This has never been absolutely demonstrated by any known method of inquiry. It is an absurdity to say that a single fibre can be traced in its continuity from side to side, seeing that the callosal fibres do not all lie in the same plane. Physiologically, there is literally no evidence to show that this mass of white matter is a means of uniting the functions of equivalent areas in the two cerebral hemispheres; and it is as yet unproved, even if it were a commissure, that unity of function would thereby result from duality of structure. In fact, the whole commissural theory of the corpus callosum has arisen simply from its lying between the two hemispheres, and from its fibres, in their middle course, seeming to run transversely, or nearly so, when roughly examined.

It is only when the brain is specially prepared for the purpose that the true destination of its fibres after crossing the middle line can be discovered. By this means there is brought out an appearance in Man and the lower animals which I have never seen figured in any work on the subject, and which careful observers, such as Flechsig, have either not observed or have ignored.

Were it true that the fibres are commissural, how is it that destructive lesions in one cortex do not affect the other? I have in my possession at present the brain of a woman in whom the first and second frontal convolutions have been completely destroyed, so far as the symptoms and morbid appearances indicate, from the time of birth, and yet the corresponding convolutions on the opposite side are quite intact. How is this to be accounted for on the commissural theory? I cannot conceive, if the one side is so intimately bound up with the other, that some mutual influence should not be exerted in a case such as this, where the deficiency has been caused at so early a period of life. Gudden's and

Monakow's experiments have shown that when certain regions of the cortex are excised in newly-born animals, the parts with which they are in connection are under-developed when the animal is fully grown. Why is it then that in the case of this large so-called commissure, if such it be, the same does not hold good? There seems to be a discrepancy here, which, until explained, should retard us from accepting as proven the usually recognised opinion of the connections and functions of the corpus callosum.

However great the difficulty may be of following the course of the callosal fibres in the adult, this in great part disappears when the embryonic brain is the subject of observation at the age I have before mentioned. Flechsig ('*Die Leitungsbahnen im Gehirn und Rückenmark*,' 1876), evidently taking it for granted that the corpus callosum is a commissure uniting different zones or areas of the cerebral cortex, has failed to notice the very remarkable appearance presented by it in from the third to the fourth month of pregnancy. He dismisses its consideration in a few words (p. 49), and says, "he will not find occasion to return to this part of the brain in the present memoir." In his succeeding publications I cannot find anything bearing on the appearances I have seen, and therefore must conclude that he has not noticed them. From this oversight I fear that his scheme of the brain, as set forth in his '*Plan des menschlichen Gehirns*' (Leipzig, 1883), requires to be reconsidered. One of the chief drawbacks of his now long-famous work on the subject of the conducting paths is that he gives in his illustrations so few drawings of the brain when cut perpendicularly. I can find only one (Pl. III. Fig. 5), and in this, unfortunately, the corpus callosum seems to have been torn. The course of the callosal fibres after they have crossed is not depicted in it, chiefly, I should think, because the brain had not been specially prepared with this object in view, but also because the child from whom it had been taken was too far advanced in development (52½ cm. long, and had lived for 2½ days). It is in a much younger foetus that the callosal fibres are to be found in their isolated state, and the reason for this is, as before referred to, that they are laid down long before

those of the peduncular tract coming down from the fissure of Rolando. Thus if the brain of a fresh human foetus about the fourth month of pregnancy be prepared by the method I employ, the corpus callosum after passing into the hemispheres can be traced with perfect accuracy throughout its entire course to its ultimate destination. Its rudimentary fibres are all deposited before the radiation of the peduncular tracts is visible, and hence it follows that if the proper age be selected, we possess in such a brain a means of studying the course and connections of the callosal fibres in their isolated state, and uncomplicated or obscured by the many other tracts that afterwards appear. The action of certain staining reagents is a great help in following out the line pursued by them.

THE CORPUS CALLOSUM IN A HUMAN EMBRYO ABOUT FOUR MONTHS OLD.

The embryo from which the following account is taken was said to be about the fourth month of intra-uterine existence. It looked as if this were true, and the state of the primary fissures in the cerebrum tended to confirm the opinion. It was received in good condition, and the brain after being hardened, or rather *toughened*, for four weeks in Müller's fluid and soaked in my freezing fluid, cut to perfection. I may mention that I have confirmed all the facts I am about to describe in many other embryos both of man and the lower animals.

The principle upon which Flechsig's work was based is that the various cerebro-spinal nerve tracts become medullated at different periods, the whiteness of the tracts which are medullated serving to distinguish them from those which are not. In former times "secondary degeneration," or the Wallerian method, was that chiefly relied upon; but as there are evidently some tracts that do not readily degenerate secondarily, a greater degree of certainty can be reached when the two methods are combined. The tracts in the spinal cord have thus been definitely laid down, and in the medulla oblongata the course of most of the nerve bundles is known. When we come to the brain, however, much still

remains to be done, the *centrum ovale* being as yet practically a *terra incognita*.

The brain of a human fœtus about this period is comparatively smooth on the surface. There are indications of the presence of the great fissures, but, as yet, the convolutions, with the exception of the island of Reil, have not appeared. Thus there is a large wide gap representing the Sylvian fossa, with the island lying exposed in it, still uncovered by the operculum. There are also indications of the first and second frontal sulci, but the fissure of Rolando is either absent or forms the slightest depression. On cutting into the brain, it is evident that the corpus callosum has advanced much beyond other parts in development. Relatively to the volume of the brain it actually appears larger than in the adult, and not only so, but the whole callosal tract can be seen passing, as I hold it does, round the ventricles down to the inner and outer capsules. This is rendered particularly evident when the brain is frozen by my method, and polished on the surface in the microtome with the section knife. The callosal tract can then be, distinctly enough, followed from the middle line to its termination.

The whole relationship of the parts, however, is rendered much more evident when a thin section is cut and stained either in solution of perosmic acid, of acid-fuchsin, or of some nuclear staining reagent such as methyl-aniline, carmine, or logwood. I shall describe the appearances presented by each of these first, and then draw certain conclusions from them.

The Perosmic-acid Preparation.—I always stain the tissue after it has been hardened—a more delicate method than that of staining the fresh tissue. I simply lay the section in a very weak solution (1 to 800) for a night, and subsequently mount it in Farrant's medium. The preparation so obtained serves both as a naked-eye and as a microscopic object, but it is most instructive as the former.

The first glance of such a preparation shows that the fibres of the corpus callosum after crossing *do not radiate* into the cerebral medulla to reach the cortex of the opposite side. On the contrary, those issuing at the side remain as a *compact ribbon-like band*, which twists upwards, outwards, and

downwards, round the ventricular cavity, and ends by entering, or rather forming, the inner and outer capsules by splitting over the lenticular nucleus. It arches much higher up in the embryo than in the adult, because the ventricle at this age is still large, and projects higher up at the sides than when the brain is fully formed. The ventricular cavity has somewhat of a Y shape, the two upper limbs corresponding to the lateral ventricles, while the lower may be taken to represent the relationship of the third. It is round the upper limbs of the Y that the callosal tract turns.

The action of the perosmic acid is to give an olive-green or light-brown tint to the whole section, but it stains the callosal tract of a dark brown, so that it becomes almost diagrammatically mapped out from all the surrounding parts. The cause of this is that *the callosal fibres are advanced in development, at this period of utero-gestation, far beyond any other nerve tract in the brain.* For while the greater part of the cerebral medulla at this time is composed simply of gelatinous tissue with embryonic cells lying in it, the callosal tract, from beginning to end, contains dense bundles of rudimentary nerve fibres.

When a small portion of the callosal tract, in a foetus of this age, is teased out and examined with a magnifying power of 450 D., the fibres of the corpus callosum have the following appearance:—They are extremely fine, and are arranged in dense bundles. In each bundle there are to be seen usually, from five or six, up to ten or twenty individual fibres. The faintest line indicates the rudimentary axis-cylinder, while round about this is a covering of finely granular material. Sometimes it happens that the axis-cylinder becomes divested of this granular sheath, and when so, its extreme delicacy becomes apparent. The granular matter is usually so abundant and so dense, that it in great part conceals the axis-cylinder.

Glacial acetic acid, however, has the effect of rendering it more apparent, but it is best seen when accidentally the granular coating has been removed by pressure. So far as I have observed, absolute alcohol and ether do not dissolve this granular sheath, but glacial acetic acid or potash both render

it much clearer even in a preparation which has been hardened. It is, therefore, in all probability not fatty as yet, and the latter reaction would seem to indicate its albuminous nature. At this stage of development I could not find any medullated fibres, and, therefore, the darkening of the tract could not be due to a fat-containing medullary sheath. The corpus callosum of an adult, when treated with perosmic acid in the same way, becomes perfectly black within 24 to 36 hours, and this is due to the large proportion of medullated fibres contained in it. The deepest stain I was able to produce in this embryo, however, was a deep brown.

The outer and inner capsules have already been referred to as being present, and it has just been stated that the callosal tract, after winding round the ventricle, forms them by splitting over the head of the lenticular nucleus. They present most of the characteristics of these structures in adult life. Thus the fibres of the inner are aggregated into bundles, which, on account of their oblique antero-posterior direction, are usually cut across in a perpendicular transverse section. Between the bundles of nerve fibres are septa of grey matter, continuous with the caudate nucleus on the one hand, and the lenticular nucleus on the other. Both capsules give a dark brown reaction with perosmic acid, the inner more than the outer.¹ There is no other part of the brain which darkens in this way, and hence my theory of the continuity of the capsular and callosal fibres gains support from this.

Not only do certain localities along the course of the tract stain deeper than others, but in the part of it which sweeps round the ventricle the perosmic acid shows, by its differential staining, that the tract consists of three layers or strata. The middle of these stains darkest, and hence I am led to suppose that it is the most highly developed. It can be traced from the middle line round to the inner capsule quite continuously. For the purpose of explanation, I shall call that part of the corpus callosum which is brought into view by separating the hemispheres, the *tectorial* part; and it can be

¹ The degree of staining depends upon the strength of the perosmic-acid solution. If a hal.-per-cent. solution be employed, the callosal tract and the two capsules become of a dark sepia brown and very opaque. A much more delicate reaction is obtained when the perosmic acid is more diluted.

readily noticed that the middle stratum forms the greater part of this. The other two strata become very attenuated as they approach the tectorial part, and finally cease to exist before the middle line is reached. The two parts of the preparation which are deepest stained with the perosmic acid are the middle stratum of the callosal tract in the tectorial part, and the inner capsule. The outer capsule is not so deeply tinted, nor is the part of the callosal tract which turns round the apex of the arch of the ventricular cavity. The staining is deeper, as before mentioned, in the middle stratum than in the other two, and on this account it can be readily traced from the tectorial part round the ventricle into the inner capsule. Few if any of its fibres seem to enter the outer capsule, which, as we shall afterwards see, appears to be more a continuation of the *upper* stratum.

The fibres, therefore, as they issue from the side of the corpus callosum, turn downwards, and form at this early period evidently the greater part, if not the whole, of the two capsules. They have been gathered in from the cortex of the opposite side, have passed through the *tectorial* part of the callosal tract, and have subsequently turned downwards. As regards the exact regions from which they arise, it is somewhat difficult to give precise details, so far as the embryonic brain at this period is concerned. The area being large, they are consequently less compactly aggregated than they are when they enter the *tectorial* part, or subsequently in their course downwards. At the same time they do not seem to be quite so highly developed as the fibres in these parts of the tract. They do not stain so deeply with perosmic acid, and when examined microscopically, they appear to be very rudimentary. A radiating arrangement is, however, distinctly apparent in the fibres coming from the cortex, most evident towards the vertex. In the adult brain a great mass of the callosal fibres comes from the vertex, specially from the margin of the longitudinal fissure. They sweep inwards with their concave side to the middle line, and it is also from this neighbourhood that the most of the callosal fibres in the embryo at this period seem to be derived.

The Acid-fuchsin Preparation.—The action of this substance

upon nerve tissues, especially those of the brain and spinal cord, is now widely known. I am inclined to believe, that we possess in it one of the most useful of all staining reagents for the purpose of tracing the course of certain nerve bundles. Its action was originally described by Weigert ('*Centralblatt f. d. med. Wissenschaft*,' 1882), and since then many workers on the normal and morbid nerve centres have been able to confirm and add to what he said of it. It differs from all other staining reagents in the fact, that apparently in the presence of a chrome-potash salt, or at any rate when the tissue has been hardened in such, it stains *the nerve fibres*, and leaves the grey matter with its cells uncoloured. I was naturally led to employ it for this brain with the view of staining the callosal tract. The particular dye is known as Acid-fuchsin "S" No. 130 of the Baden Aniline manufactory, and the sample I have of it I procured from Dr. Grüber, of Leipzig.

It was questionable whether, with the rudimentary fibres of the corpus callosum, the same reaction would be given as with those which were fully developed. I found, however, that the callosal tract in the embryonic brain stains, if not so differentially as in the adult, yet so much more deeply than other portions of the cerebral medulla, that it forms to the naked eye a pink-coloured band, as clearly drawn out as if it had been done with a camel-hair pencil. The whole tract and the two capsules give this reaction, so that the continuity of the one with the other is rendered extremely evident. Microscopically it is seen to be due to the coloration of the rudimentary fibres, not to that of the nuclei which are abundantly interspersed between them.

As, however, methyl-aniline gives, if anything, a still more differential picture when the preparation is examined microscopically, I shall describe the minute appearances as seen in one stained by this latter substance.

The Methyl-aniline Preparation.—Flechsig has pointed out, that when a tract begins to become medullated, the nucleated cells within it increase greatly in number, and if such a tract be treated with a nuclear staining reagent, the coloration forms a trustworthy guide to its position, both as a naked-eye object and microscopically. The same nucleated cells are seen in

abundance in the callosal tract at this age, and when stained they form an important indication of the course of its fibres. Several nuclear staining materials bring out like appearances. The one I have found best is methyl-aniline. Logwood does very well, but the former is more delicate and differential, and various degrees of staining can be obtained with it better than with logwood. I overstain the preparation, and wash out to the proper degree with dilute acetic acid and alcohol. The preparation is mounted in Farrant's solution, as previously.

The callosal tract is again seen to consist of three layers; but there is this difference in regard to their staining, that the uppermost has stained deepest, the lowest least so, and the middle presents a medium amount of coloration. The lowest layer appears to be purely cellular, the cells are all rounded, and they possess large nuclei which stain vividly with the methyl-aniline, more intensely even than those of the cortex. It measures from a quarter to a half a millimetre in thickness. Its course is to be traced from the tectorial part entirely round the ventricle, and continuously downwards to the rudimentary caudate nucleus. It is a remarkable fact, that the nearer the caudate nucleus is approached, the more intense the staining becomes, until in the caudate nucleus itself a distinct reaction is produced by this dye, the colour of the cells contained in it being a bright blue. Near the septum lucidum the coloration is much less intense, and the layer becomes so much attenuated that it is gradually lost before reaching the middle line. The same intense affinity for staining media characteristic of the cells contained in this layer is seen when logwood is employed, but to a minor extent, methyl-aniline, as just mentioned, giving a blue *reaction* with its cells. I could not distinguish any fibres within it even of a rudimentary character, nor could I find at any part fibres approaching it as if about to become incorporated with it. Its cells seemed to pass continuously into those of the still rudimentary caudate nucleus. What this layer represents I cannot definitely say. In the *adult* brain, there are always seen in this part of the corpus callosum a few bundles of fibres, turning sharply downwards and inwards to the caudate nucleus. Some of them enter it, and I think it

quite possible that, later on, this layer becomes converted into these fibres, and so still retains its primary connection with the caudate nucleus. I shall not commit myself, however, to this view, until further inquiring into the matter.

The caudate nucleus at this period is entirely cellular. Fibres cannot be seen entering it from the inner capsule, and the cells are so closely packed, that when stained as described it looks like a uniform intensely blue mass. Lying outside of it, between it and the inner capsule, is a little acutely oval-shaped cellular piece of tissue, much less intensely stained, but composed of the same kind of cells. It is present in all the sections from this neighbourhood; and were it not for the sharp differentiation brought out by the staining, I should have included it simply as part of the caudate nucleus. It sends processes between the bundles of fibres of the inner capsule, and is very vascular. It stains exactly in the same manner as the lenticular nucleus, and the processes or septa which run in between the inner capsule bundles unite it directly with this nucleus.

Passing now to the middle stratum of the callosal tract, it is seen to be composed in the tectorial part of dense masses of fine fibres, with only a few nucleated cells between them. It measures from one to one and a half millimetres in thickness. Tracing it along the callosal tract, the same characteristics distinguish it until it reaches the vault of the ventricular cavity. Here, just as the tract begins to turn round, brightly stained nuclei become visible in it, and they continue until the callosal tract divides into the outer and inner capsules. In these the nucleation almost entirely ceases. The nuclei are surrounded by a little very delicate protoplasm which does not stain, and the whole cell, as a rule, is not larger than a leucocyte. They are distributed in rows between the nerve fibres, so that in a preparation such as this in which they are brightly stained, they indicate in a very demonstrative manner the course followed by the bundles of delicate nerve fibres. When more highly magnified (350 D.), they are found to exactly correspond to the inclination of the several nerve bundles.

Tracing this middle and largest stratum of the callosal tract

downwards after turning round the ventricles, it is evident what becomes of it. It splits into the outer and inner capsules over the head of the lenticular nucleus. Not only does it split into these, but at this period of development *the entire inner capsule and a great part of the outer are formed by it*. After the most careful search I cannot find any other system of fibres going into the capsules—and yet they are perfectly distinct, and appear quite as advanced in development as any part of the callosal tract.

This, I hold, entirely coincides with the view announced by me in my communication to the Royal Society. I there stated, that I believed the anterior limb of the inner capsule to be composed almost entirely of crossed callosal fibres. Certainly some bundles enter it from the first frontal convolution of the same side, and probably other tracts may be derived directly from the tip of the frontal lobe, such as Meynert's anterior peduncle of the thalamus. This, I grant, may be true, but what I still uphold as entirely borne out by the examination of the embryonic brain, is that the main bulk of the fibres of the anterior limb of the inner capsule is callosal.

The most superficial of the three layers of the callosal tract is also, in the brain I am describing, the most highly nucleated. It is seen to arise in a very attenuated row of cells, which appears to be continuous with a delicate cellular layer lying on the upper surface of the tectorial part. It measures from half to one-and-a-half millimetres in thickness. Starting then with this attenuated internal extremity of the most superficial of the three layers of the callosal tract, it is seen rapidly to increase in bulk outwards, until opposite the highest point of the ventricular cavity, its maximum of breadth is obtained. It here forms a pyramid-like projection, and the particular shape thus imparted to the arch of the "crossed callosal tract" persists in some regions of the brain in adult life, more particularly in the parietal. As a rule, however, the pyramidal shape is lost when the brain is fully developed, so that the arch of the "crossed callosal tract" becomes in the adult more or less rounded. Traced further downwards, it is seen that the upper stratum becomes incorporated with the middle, and I think that most of its

fibres ultimately pass into the outer capsule. It is intensely nucleated throughout, and between the rows of nuclei fibres less advanced in development than those of the middle stratum are contained.

The pyramidal projection formed by this stratum is easily accounted for when examined microscopically. The fibres and their corresponding rows of nuclei are tilted upwards at this portion of the tract, and are much more acutely arched than in any other part. It looks as if they were driven into this position by the ventricle, the pyramid-like contour of the tract corresponding in shape with the apex of the vault of the cavity.

Such being the composition of the callosal tract at this age, an interesting question comes to be whether there are any signs as yet of the peduncular tract, described and figured by Flechsig, in a viable foetus of $52\frac{1}{2}$ cm. long (*Die Leitungsbahnen,* &c., Pl. III. Fig. 5, *c.c.*), and which one can see with little difficulty even in an adult brain when properly prepared. It will be noticed from the drawing he gives of it (naked-eye appearance only) that it comes down from the margin of the longitudinal fissure. He describes it thus at page 29:—"I found," he says, "in the medulla of the hemispheres a white mass which deserves minute examination, both on account of the time of its appearance, and of its special morphological properties. It was apparent in frontal sections which ran through the upper parietal region opposite the meeting-point of the two central convolutions, more especially the upper and anterior part of the posterior. It consisted of a small sharply demarcated white streak, which, leaving the external part of the capsule, that is to say, the neighbourhood of the divisions of the lenticular nucleus, ran first somewhat outwards, curved for about $\frac{1}{2}$ cm. above the ventricle, was then sharply bent inwards, and finally passed upwards to end in the part of the posterior central convolution, which lies nearest the fissure of Rolando. While this band hardly reached up to the cortex in a $2\frac{1}{2}$ days old child, it passed up to within $\frac{1}{2}$ cm. of the same in one 9 days old."

In my communication to the Royal Society on the subject of the corpus callosum, I stated that the "corona radiata," as

usually described, does not exist. The common idea is that the fibres of the inner capsule coming up from the peduncle and basal ganglia, all radiate outwards into the cortex of the same side. That is to say, the fibres simply spread from the inner capsule in all directions, and become attached to the hemisphere on the same side.

With this view I cannot agree, and the more I work at the matter, the more I become convinced that it is erroneous. Flechsig, in the passage I have already quoted, traces a thin white medullated band from the inner capsule to the upper part of the fissure of Rolando, in the neighbourhood of the paracentral lobule. This appears in the foetus when viable, and almost at the full time of utero-gestation. As he figures it (Pl. III. Fig. 5, *c.e*), the band appears very slender—a mere twig compared with the enormous mass of the inner capsule or centrum ovale. He shows that this is directly continuous with the inner capsule, and that it comes from that region of the cortex which corresponds to the upper part of Ferrier's motor area. It further is equivalent in bulk, it will be admitted, to the tract which degenerates in an old destructive lesion in this neighbourhood, and hence there is little doubt that it consists of fibres which pass directly from the pedunculi upwards. He does not say, however, that the remainder of the so-called *corona radiata* can be traced in the same way from inner capsule to cortex. No doubt certain special tracts, such as Gratiolet's band to the occipital lobe, can, but the enormous mass of the *corona radiata* as described cannot be directly followed out in the same way. The band going to the motor area, as above described, forms a mere fraction of the fibres of the inner capsule, and it alone can be distinctly traced in its development from the inner capsule to the cortex of the same side.

Now in any adult brain these direct fibres can be readily seen when prepared in the method formerly described by me ('BRAIN,' July, 1883). They come from the margin of the longitudinal fissure, are aggregated into comparatively coarse bundles, and finally enter the inner capsule. I trace them forwards in the adult brain much further than Flechsig does in the embryo. I do not think that they are derived merely

from the margins of the Rolandic fissure, or from the paracentral lobule; for I find them also coming down from the first frontal posteriorly. They are certainly, however, most abundant at the line of the Rolandic fissure, and the bundles here are coarser than elsewhere. They seem to be entirely confined to the inner capsule in their progress downwards. That some of them also enter the outer capsule I am not prepared to deny, but it has always seemed to me that the outer capsule, in its inner half at least, is composed of crossed callosal fibres derived from the "crossed callosal tract."

In the fœtus of four months, however, the inner and outer capsules are well formed and filled with developing nerve-fibres, while the band running up to the margin of the hemisphere as described by Flechsig is non-existent. The capsules, further, can be distinctly traced continuously into the corpus callosum along the callosal tract, as I have just described. Here then is a system (the callosal) well developed before any direct fibres from the cortex of the same side have made their appearance. The two capsules are of large size—relatively to the bulk of the brain of very large size, and they correspond in dimensions to the bulk of the callosal tract. If then the "corona radiata" of the inner capsule sends its fibres to the cortex of the same side, how is it that where the "corona radiata" is not present, the two capsules are yet laid down with the shape and features which characterise them in adult life? The band described by Flechsig as coming down from the motor area is one of the best marked of all the systems of the so-called "corona radiata." Here, however, in this four-months-brain there is not a trace of it, while the bundles of fibres in the two capsules are perfectly distinct. The fact, that the callosal tract can be traced macroscopically and microscopically to enter the capsules in the brain of this age, affords, I think, the explanation; and this explanation I hold lies herein, that not only in fœtal, but in adult life, the inner and outer capsules are largely composed of fibres which are callosal in their derivation. As development goes on from four months upwards, *direct* bands undoubtedly pass into them from various parts of the brain. These, however, form a small part of their bulk, compared with those of callosal origin. I

think, therefore, without drawing any very detailed conclusions from the foregoing facts as seen in the four-months-brain, it may fairly be granted that these facts undoubtedly afford strong support to the views expressed by me in my communication to the Royal Society.

In summing up the main conclusions that may be inferred from the study of the four-months-embryo, I would say that they all tend to refute the idea of the corpus callosum being a commissure, and to support that of its being in reality a decussation of *certain* of the cortical fibres. These arising from the cortex on one side, pass through the tectorial part of this body to the opposite, and after circumventing the ventricular cavity, turn downwards to form the greater part of the inner capsule, at least in its anterior limb, as well as the inner half of the outer capsule.

If this then be the course of the callosal fibres in the middle part of the cerebrum, what is the disposition of those which take their origin from the frontal tips? The usual idea, as every one knows, is that the callosal fibres coming from the one frontal tip, pass through the genu of the corpus callosum, to gain a corresponding situation in the opposite tip. In this course they are supposed to run horizontally. I have long been persuaded that this idea is erroneous, but it was not till I undertook the present inquiry on the foetal brain that I could actually demonstrate it. The course pursued by the callosal fibres in this part of the brain, is a much more difficult matter to demonstrate in the adult than might be supposed. It might be thought that the track of a large mass of fibres like this could be easily followed up. No greater error could be made. The intertwining of different systems is so complex, that I believe it is only in the foetus, where the callosal system is alone developed, that it is to be clearly made out.

The reason for the peculiar arched course of the callosal tract, as described, is obvious. It is to allow the fibres to circumvent the lateral ventricle, and if my idea as to the course of these fibres be correct, the same obstacle has to be overcome by those fibres derived from the extreme anterior frontal region. What I have found both in the foetus and the

adult is that, instead of the callosal fibres derived from the anterior frontal region arching between the frontal tip of one side and that of the other, the fibres coming from, say, the left side, pass back horizontally to the genu corporis callosi, cross in this more or less obliquely, and, gaining the right side, turn ultimately backwards into the inner capsule. Now the anterior horn of the lateral ventricle lies considerably further forwards than the genu of the corpus callosum, and hence, in order to circumvent this obstacle to their gaining the inner capsule, they have to follow a circuitous course around it. When I found that the callosal tract was so well defined in perpendicular sections of the above-described foetal brain, it immediately struck me that if my theory were correct, a *horizontal* section made through the tip of the frontal lobe ought to show the callosal fibres turning round the anterior horn of the ventricle, in order to enter the inner capsule. This proved to be actually the case, far beyond my expectations. A band quite as distinct as that seen in perpendicular sections is to be found in a foetus of this age in horizontal sections, it runs from the corpus callosum forwards, outwards, and backwards, around the anterior horn of the ventricle. In the adult brain this band becomes so interwoven with other medullated fibres that it cannot be distinctly demonstrated.

As regards the future course of these fibres I cannot say anything further at present, as it would require a great wealth of illustration to make what I should like to say on the subject comprehensible. The main fact that I would wish to record in this brief communication is that which has been chiefly dwelt upon, namely, that the examination of the embryonic brain supports the conclusion I formerly arrived at of the corpus callosum being not a commissure, but in reality a decussation similar to that of the anterior pyramids.

ON SOME OF THE RARER FORMS OF MUSCULAR ATROPHIES.

BY J. DRESCHFELD, M.D., F.R.C.P.,

*Professor of Pathology, Victoria University; Physician to the Royal
Infirmary, Manchester.*

THERE is scarcely any subject of the whole of neuropathology which presents such a large field for observations and enquiries as the chapter on muscular atrophies. Thanks to many important investigations, we are beginning to understand more clearly, and classify more correctly, the many varied forms of diseases characterised chiefly by trophic changes with more or less paralysis in the muscles.

Much, however, remains yet to be done, and many gaps will yet have to be filled up, before the pathology of the different forms of muscular atrophy may be said to be definitely settled. If, in spite of the numerous recent observations, there still remain many points of dispute, the reasons are not far to seek; many of the incurable cases run a very chronic course, and do not give us the opportunity of a pathological examination; others undergo marked improvement, and even complete cure, and thus also escape the pathologist; while many, again, though they may differ materially both as regards the nature and localisation of the disease, present a certain similarity in the symptoms. As a small contribution I beg briefly to relate some rarer forms of muscular atrophy which I have observed during the last few years.

In classifying muscular atrophies we may still be guided, though with certain restrictions, by the principle laid down by Waller, that the spinal cord exercises a trophic influence over the motor nerves and muscles, and that the motor ganglia-cells in the anterior horns form the trophic centre. Lesions of the brain, therefore, as long as the spinal trophic centres are unaffected, will not produce muscular atrophies, and when such

muscular atrophies result or are associated with brain-lesions, we have either, as in the cases recorded by Pitres, marked atrophy of the motor ganglia-cells, or, as in the case reported by Kojeronikoff,¹ a degeneration of the whole of the pyramidal tract propagated to the motor ganglia-cells in the cortex of the brain.

A number of atrophies will thus result from a primary affection, either inflammation or degeneration of the large motor ganglia-cells. To this group belong, as is well known, infantile paralysis and the acute poliomyelitis of adults; progressive muscular atrophy; subacute and chronic polio-myelitis and amyotrophic lateral sclerosis, and the corresponding affections of the nuclei of the motor cranial nerves, namely acute and chronic bulbar paralysis (glosso-labial pharyngeal paralysis and Wernicke's polio-encephalitis superior, acute and chronic), which seems to correspond to the ophthalmoplegia of Hutchinson and Mauthner.

More recent observations have taught us to separate from this group several types which formerly were classed with it; thus from progressive muscular atrophy we must separate Erb's juvenile form, chiefly characterised by the limitation of the affection to the muscles of the shoulder girdle and arms, and in some cases the thigh, while the forearms and hands remain free, by the absence of fibrillar twitchings, and by the absence of degenerative reaction; another form is that described by Charcot and Déjérine, where the facial muscles are the first to be affected. Again, many cases of chronic poliomyelitis are now looked upon as belonging to the group of peripheric neuritis, and even the amyotrophic lateral sclerosis is looked upon by some, we think wrongly, as a form of progressive muscular atrophy.

(Within the last six years, two cases showing during life the typical symptoms of amyotrophic lateral sclerosis have occurred in the practice of my colleague Dr. Leech, in the Manchester Infirmary; and in both cases the microscopic changes of the spinal cord, and also in the medulla, showed the well-marked affection of the motor-ganglia cells with sclerosis of the lateral columns. As both cases will be

¹ 'Arch. de Neurol.,' 1884, No. 18.

published by Dr. Leech, I need not dwell on them further here.)

Besides the primary affection of the motor cells, there are some other spinal diseases giving rise to extensive muscular atrophies, and where the motor-ganglia cells are chiefly concerned in the disease process; to this group belongs the perpendymary myelitis of Hallopeau, the syringo-myelia and the diffuse glioma (of which the latter two have been more recently studied), and the rare cases of hæmorrhage into the spinal cord.

In other affections of the spinal cord, such as locomotor ataxy, tumours of the cord, syphilitic diseases of the cord, the ganglia-cells may be implicated secondarily, and muscular atrophies of great extent may ensue.

In a second great group of muscular atrophies the spinal cord is found normal, and the affection resides chiefly in the peripheric and intermuscular nerves, thus causing separation of the muscles from their trophic centre, and hence muscular atrophy. Our knowledge of this affection, as far as it concerns the idiopathic non-traumatic forms, is of recent date, and we may now include the multiple peripheric neuritis of Leyden, lead paralysis with atrophies, alcoholic paralysis with atrophies and some at least of the paralyses and atrophies, following diphtheria, typhoid and other zymotic diseases and the epidemic disease, Beri-Beri, besides the secondary atrophies, due to wounds of nerves, pressure by tumours, lepra, &c. As intermediate between the first and second group, we may consider the atrophies following affections of the membranes of the spinal cord, notably those produced by pachymeningitis.

In a third group neither cord, nerve root nor peripheric nerves are affected, and the affection is a primary myotic. Besides the juvenile form of Erb, and Charcot, and Déjérine's form, this group also includes pseudo-hypertrophic paralysis.

(It has been held by some, that probably some of the atrophies of this group of affections may have their starting-point in the terminal muscle plates; as yet we have no observations on man, and considering the difficulty of such investigations, this is not to be wondered at. Gessler¹ has

¹ 'Die motorische Endplatte;' Leipzig, 1885.

quite recently published some observations on changes in the terminal muscle plates observed after section of peripheric nerves in animals. He found changes mainly in the granular nuclei of these plates. As these changes were found along with the changes in the peripheric nerves, it cannot be said that our knowledge of the myopathic atrophies has thus been in any way advanced. Nevertheless the subject is one which, no doubt, will receive further attention.)

Lastly we have reflex atrophies, of which those studied by Charcot,¹ and associated with joint-affections, are the best known. In these atrophies it is assumed that the trophic ganglia cells in the cord are irritated in a reflex way by means of the nerves of the affected joint. (In some of the forms of atrophy, as, for instance, in lead atrophy, de Watteville and others believe that the ganglia cells are implicated, though they may not show any changes when examined microscopically.)

In spite of our different subdivisions, it will still be found that the clinical pictures of the several forms vary but slightly, that there are yet some forms which do not exactly fit into any of the given groups, as will be seen from some of the cases described below. In describing the cases which have come under my observation, I will group them, as far as possible, according to the classification just given.

The cases which I have observed of progressive muscular atrophy (Aran Duchenne's type) have been of the ordinary character, with the exception of two which have been under observation for many years, and differ from the ordinary type in two important particulars; namely the affection, though commencing slowly, remained limited to the small muscles of the hands, and has now continued stationary for several years; the atrophy was associated with extensive analgesia and loss of the sense for temperature; whilst the common tactile sensibility, the sense for weight, and the muscular sense remained normal. Of these two cases I will give one more fully, the second resembling this in almost every particular.

¹ 'Progrès médical,' 1882.

CASE I.

A. G. has been under observation since 1878, when he was admitted first as an out-patient and then as an in-patient into the Manchester Infirmary. On his admission, Nov. 19, 1878, the following notes were taken: Pat., æt. 23, mechanic; the father is living and healthy; the mother and one sister died of consumption; has two sisters living, who are in perfect health. The patient himself has enjoyed good health till a year before admission; has been regular in his habits, and has not suffered from rheumatism or syphilis. Twelve months before admission he noticed his fingers getting cold and numb, and found that he could not execute finer movements, such as picking up a pin; the weakness gradually increased, and he lost power in both hands; he often noticed scratches and burns on his hands without being able to account for them, and without feeling any pain.

Condition on Admission.—The patient looks healthy, is somewhat spare, and of good intelligence. The examination of the thoracic and abdominal organs reveals normal relations. Pulse 70, regular and fairly strong, temp. normal; skin moist; bladder and rectum unaffected. As regards the nervous system, there are no cerebral symptoms, and no symptoms on the part of any of the cranial nerves; the lower extremities are normal as regards motion, nutrition and sensibility. *Upper Extremities.*—The hands and forearms are seen to have lost a considerable amount of flesh; the small muscles of the hand being especially atrophied; the thenar and hypothenar eminences have completely disappeared; the interossei are considerably atrophied, and both hands present the “main en griffe.” The flexors of the fingers, the pronators and the supinators act fairly well. The muscles of the arm, shoulder, neck and trunk are perfectly normal as regards motion. The left hand is slightly more affected than the right. No fibrillar twitchings in any of the muscles.

The tactile sensibility is normal, and the patient can, with his eyes shut, tell correctly the outlines and physical condition of the surface of any object he touches. There is, however,

complete analgesia of the fingers and thumbs, and of the palm and back of hand; it extends also in front, behind, and on the outer side of each forearm quite up to the elbow, while on the inner side the analgesia ceases about two inches below the elbow. There is, however, diminished sensibility for pain along the whole of both arms and shoulders, reaching to the outer end of the clavicle in front and nearly up to the spine of the scapula behind. The whole of the analgesic area shows complete absence of sense of temperature; the patient is quite unable to distinguish between hot and cold. The patient has often burnt his fingers in handling burning coals; he has felt no pain whatever, even though the burn resulted in an ulcer (which, however, healed very readily). The sense for weight, tested in the usual manner, was found to be fairly normal. The patient knew also the exact position of his fingers or hands, when tried in various ways with the eyes shut.

The superficial and deep reflexes are normal in the lower extremities; no tendon reflexes in the upper extremities.

MEASUREMENTS OF THE AFFECTED PARTS.

Right side: over styloid process	6½ inch.
1 inch above styloid proc.	6¼ "
1½ inch below olecranon	9½ "
Left side: over styloid	6½ "
1 inch above styloid	6 "
1½ inch below olecranon	9½ "

The hands feel cold; their temp. is 95. The parts to which electricity has been applied show a dusky red appearance for a long time after the application has ceased.

Electric-reactions.—With the faradic current it was found that, with the strongest current, no contraction could be produced in the interossei muscles and muscles of the thenar and hypothenar eminences whilst the other muscles reacted fairly well to the faradic current.

Galvanic Reactions.—Dixon Mann's battery. Direct muscular excitation:

Right hand, dorsal interossei	..	KCC	40 cells	20°
		KOC	none.	
		ACC	20 cells	15°
		AOC	40 "	28°

Flexors of Fingers	KCC	15	cells	20°
			KOC	40	"	28°
			ACC	15	"	20°
			AOC	20	"	22°
Extensors of Fingers	KCC	15	"	20°
			KOC	35	"	25°
			ACC	15	"	15°
			AOC	25	"	20°

The left side gave very similar results.

Galvanisation of Nerves :—

Ulnar (right side)	KCC	8	cells	10°
			KOC	20	"	20°
			ACC	30	"	22°
			AOC	30	"	20°

These numbers show degenerative reaction in the affected muscles.

The patient was treated after Dr. Morgan's plan by direct application of electricity (faradic and galvanic current) to the muscles; fine needles were inserted into the affected muscles, and these connected with one pole of the battery; while the other pole was applied to some indifferent point, or sometimes the two poles were connected with two needles, both inserted into the same muscle. For a time it seemed as if the patient improved, but no permanent improvement resulted, and the patient was discharged in March 1879, very much in the same condition as on admission. Since then the patient has presented himself many times, and the disease has made in the last seven years but very little progress. When examined a few months ago, it was found that, as regards the atrophy of the small muscles of the hand, there was little change; the patient stated that the wrist felt weaker, and certainly the forearms had become slightly thinner, as shown by the following measurements :

Right side: over styloid process		6½	inch.
1 inch above styloid		6	"
1½ inch below olecranon		9¼	"
Left side: over styloid		6½	"
1 inch above styloid		5¾	"
1½ inch below olecranon		9¼	"

The complete analgesia was limited the same as on admission; but the diminished sensibility to pain observed

before on arms and shoulder had now extended transversely across the chest and back. On the chest the upper limit corresponded to a line a little above the clavicle and its lower limit on the left side to the sixth rib, and on the right side to the fifth rib. On the back the area of diminished sensibility was limited, above by a transverse line at the level of the sixth cervical vertebra, and below by a similar line opposite the fifth dorsal.

The electric examination gave the following results :—

Faradic Current :—

Right side (upper extr.) :

Extensor digitor. 120 mm.

(Du Bois-Reymond's apparatus.)

Flexor carpi ulnaris 125"

Left side (upper extr.) :

Extensor digit. 110 mm.

Flexor carpi ulnaris 120 ..

Both on the right and left side no reaction even with the strongest current in the lumbrici, abduct. minim. digiti, flexor brevis minimi digiti, abductor pollicis, flexor brevis pollicis and opponens pollicis.

Galvanic Current (direct muscular) :—

Right side: Extensors ♂KCC 20 cells.

♂ACC 30 ..

Flexors KCC 25 ..

♂ACC 30 ..

Left side: nearly the same as right.

In the atrophied muscles, by applying both poles to the muscle it was found that even 50 cells produced no KCC, while ACC was slightly noticed with 50 cells and several times even 45 cells.

CASE II.

The second case resembles the case just given very closely.

Martha D., admitted September 1879, æt. 33; married; has four living children, all healthy; has been engaged in mill-work, but left off work long before her affection commenced. She had always enjoyed good health, and only complained of occasional cough. About four years before date of admission

she noticed that she could not grip things so well with her hands, or hold objects properly with her hands; also noticed her hands and forearms getting weaker and thinner. She never experienced any pain in her hands, but was struck with the numbness of her fingers. The numbness she began to notice about three years ago, and for the last two years her fingers have been bent, and she has not been able to stretch them or use them for her household work.

On admission of the patient, it is found that her troubles are entirely limited to the hands and forearms. There is marked atrophy of the small muscles of both hands, and marked "main en griffe." The forearms are also somewhat atrophied, though the flexors and extensors of fingers, the pronators and supinators, show considerable power of motion.

The measurements gave the following results:—

Right side:	above wrist	5 $\frac{1}{2}$ inch.
	below elbow	8 "
	1 inch above elbow	8 $\frac{1}{8}$ "
	belly of biceps	9 "
Left side:	above wrist	5 $\frac{1}{2}$ "
	below elbow	8 $\frac{3}{4}$ "
	1 inch above elbow	8 $\frac{7}{8}$ "
	belly of biceps	9 $\frac{1}{8}$ "

There is marked analgesia of the whole of the hand and forearm, nearly up to the elbow on both sides; on the right side the anterior, posterior and external side of arm, almost up to the shoulder, is analgesic, there is also complete loss of sensibility for temperature over the analgesic part; whilst the common tactile sensibility is perfectly normal.

As regards the electrical reactions, the observation is unfortunately incomplete. The faradic current only was tried, and it was found that the atrophied muscles did not respond even to the strongest current, while the non-affected muscles responded readily. No other part of the muscular or nervous system was found affected; bladder and rectum were likewise unaffected, and the examination of the internal organs showed nothing abnormal, except the presence of bronchitis.

The patient was also treated by the subcutaneous application of electricity, without, however, any material benefit.

I saw the patient again about three years ago, when she was

very much in the same condition as on her admission to the Infirmary. The atrophy had certainly not progressed much, and the analgesia was as well marked as then.

Cases of progressive muscular atrophy in which spots of analgesia occur do not seem to be very uncommon; but I have looked for published cases like the two given above, where there was no anæsthesia, but marked analgesia, and I cannot find any other except the one recorded by Mosler and Landois,¹ which closely corresponds to them.

CASE III.

The next case I wish to place on record is one of chronic poliomyelitis, and the publication of this case appears to me to be especially opportune, as some seem to doubt the existence of this disease, believing that many of the recorded cases are cases of peripheric neuritis. There are already some authentic and well-observed cases on record² with post-mortem records; the most interesting case, however, is the one recorded by Landouzy and Déjérine,³ which, though more acute in its onset, belongs yet to this type. In one of the two cases observed by these authors, the patient completely recovered from his spinal symptoms (the abolition of the tendon-reflexes remaining as the only symptom), and died subsequently from pulmonary tuberculosis. The spinal cord, however, showed marked alterations of the anterior grey matter along its whole length, while the anterior roots and peripheric nerves showed no appreciable alterations.

The case which I have to report had been under observation from its commencement to its fatal termination, and the symptoms during life consisted of a gradual paralysis, with marked atrophy, first of the lower extremities, then of the muscles of the trunk, and then those of the upper extremities; the medulla remaining free to the end. There were no sensory troubles, and bladder and rectum remained normal. The tendon-reflexes in the lower extremities were absent, while

¹ 'Berl. Klin. Wochenschrift,' 1868, p. 458.

² See, as regards literature, Ross, 'Diseases of the Nervous System,' 2nd ed. vol. i. p. 912; and Erb, 'BRAIN,' 1882.

³ 'Revue de Méd.' 1882, Nos. 8 and 12.

those of the upper extremities became marked towards the end. The post-mortem examination showed the affection limited to the anterior horns, except in the cervical and upper dorsal region, where slight sclerosis of the lateral columns was observed. The nuclei in the medulla were found healthy; there was, however, found a small recent hæmorrhage in the nucleus of the pneumogastric, which probably accounted for the marked dyspnœa during the last days of the patient's life and the somewhat sudden termination, together with some other slight changes in the medulla.

The following is a brief abstract of the case :—

Wm. S., admitted into the Infirmary, Dec. 2, 1881; died June 16, 1884.

Soon after admission the following notes were taken: *Æt.* 36; minder in a mill; married; no children. Father and mother died when patient was still very young.

The patient had been employed in a cotton mill for twenty-six years, and had always enjoyed good health; he had been abstemious in his habits, a moderate smoker, and had never had any venereal disease.

In March 1881, while at work, he had a fainting fit; but this passed off without leaving any trace, and he was able half an hour after the attack to walk home, and went to his work the next day. Some weeks after the attack he noticed a gradual weakness of his right foot and leg, and he found that on walking he had to drag the right leg slightly; he also noticed the right leg to get thinner and flabbier. He, however, never experienced the least pain.

On admission, the patient presented a healthy appearance, was well-nourished and of ruddy complexion. The circulatory, respiratory, and chylopoietic system appears normal; the urine has sp. gr. 1020, is acid, and contains neither albumen nor sugar.

The brain, special sense-organs and cranial nerves are normal; likewise the muscles and nerves of the trunk and upper extremities. The left lower extremity shows no changes relating either to the motor, sensory, or vaso-motor nerves, and the only changes observed relate to the right lower extremity.

The patient is unable to perform dorsal flexion and adduction of the foot and toes; the foot cannot be abducted, and the arch of the foot is flattened; the patient is able to flex and extend the knee, also flex and extend the right hip very well—perform adduction, abduction and rotation of the right lower extremity with ease; yet all these muscles on the right show, when tested, less power than the corresponding muscles of the left lower extremity. The muscles on the right side feel flabby, and there is considerable atrophy, as shown by the measurement.

RIGHT.			LEFT.
Mid-calf	11½	inch.	12½ inch.
2 inches above knee	13	..	14½ ..
Mid-thigh	15	..	17½ ..

The sensibility is perfectly normal both to touch, pain, temperature, and pressure. The muscular sense is intact.

The patient's walk shows the characteristic appearance of the paralysis; the right foot hangs down and is not raised from the ground; in taking the steps the knee is flexed and the foot thus raised, which falls down again at the next step in a flaccid condition.

The right patellar tendon-reflex, which was still present on admission of patient, but diminished when compared to the left, soon disappeared altogether.

On testing the electric relations of the muscles of the right leg, it was found that the tibial. antic., the peroneus and extensor longus digit. scarcely answered to the strongest faradic current; the gastrocnemii, flexor long. digit. and tibial. anticus answered better. (The galvanic reactions were not tried till later.)

The patient left the Infirmary on March 8th, 1882, very much in the same condition as on admission. He went to the Convalescent Hospital at Cheadle for some time, and then went home, when he became gradually worse.

He was readmitted on Feb. 16, 1883, when his condition was found very much worse, as will be seen from the following report, taken shortly after the patient's readmission.

The patient lies in bed, and cannot rise from the recumbent posture without help, from weakness of the muscles of the

back; but, sitting up, he can easily straighten his back by contraction of his lumbar muscles.

There is only slight hardening of the recti abdominis on their contraction.

Similarly he cannot get from the sitting to the recumbent posture gradually, but goes down with a bump. He is just able to stand with support, but is quite unable to walk. The superficial (cremaster, abdominal, epigastric and scapular) reflexes are slightly marked.

Lower Extremities.—The right leg and thigh is completely paralysed, and the muscles considerably wasted. There is not the least rigidity about the joints, and the extremity remains in exactly the position in which it is placed. The plantar reflex is absent, likewise the patellar tendon-reflex. Sensibility (in its various kinds) of the right leg is normal. Patient complains of coldness of the limb.

The left leg is also paralysed and atrophied, but not to the same extent as the right. There is paralysis of the extensors of the pons and of the peronei; there is also some paresia of the extensors of the toes and of the hamstring muscles, but the muscles of the calf and the quadriceps extensor are little affected. There is paresis of the flexors of the thigh on the abdomen. The plant or reflex is absent. Patellar reflex present, sensibilities normal.

MEASUREMENTS OF LOWER EXTREMITIES:

	RIGHT.	LEFT.
Calf	10½ inch.	10¾ inch.
Mid-thigh ..	14 ..	16 ..

On striking the muscles of either lower extremity, fibrillar twitchings are seen.

Electric reactions.—The faradic current elicits no contraction whatever in any of the muscles of the right lower extremity. In the left limb the extensors of the foot and toes, the peronei, the muscles of the calf, the extensors of the thigh and the hamstring muscles, responded slightly to strong currents.

Galvanic reactions: Right leg.—The extensors *brev. digit.*, the peronei, and the extensors of foot do not contract at all.

Calf muscles :—

KCC	15 cells	10° slight contr.
KOC	45 „	25° (doubtful contr.)
ACC	10 „	15° marked.
AC Tet.	..	25 „	25°
AOC	20 „	— (very painful.)

Left leg :—

Extens. long. digit. and tibialis antic.

KCC	20 cells	13°
AC Tet.	..	20 „	15°

Extens. brev. digit.:

KCC	15 „	10°
ACC	10 „	10°
AC Tet.	..	25 „	18°

Peronei :

KCC	15 „	10°
ACC	10 „	10°
AC Tet.	..	20 „	18°

Calf muscles :—

KCC	15 „	7°
ACC	10 „	8°
AC Tet.	..	20 „	15°

The upper extremities, muscles of neck and head appear perfectly normal on admission. All the internal organs appear healthy. There is no affection of either bladder or rectum.

During his further stay in the hospital he gradually grew worse, and the muscles of the upper extremity commenced to be affected, the shoulder arm and muscles being the first to show signs of paralysis and atrophy. I will briefly quote from the case-book the condition of the patient on Sept. 14, 1883. The patient lies in bed; can neither stand nor walk; can only assume the sitting posture with assistance, and he has great difficulty to remain in this posture. The right lower extremity is quite paralysed and atrophied, the joints flaccid. The left lower extremity is now almost completely paralysed, with the exception of the extensors of the leg on the thigh; the muscles of the left lower extremity are likewise flaccid; the patellar tendon-reflex on left side has now also disappeared. Sensibilities intact.

Upper extremities.—There is weakness of all the muscles, compared with their previous condition, but the shoulder and arm muscles are most affected. The flexors and extensors of

wrist and small muscles of hand act fairly well, and patient can perform all the movements with his fingers well. He can pronate and supinate freely with the left forearm, but is unable to do so fully on the right side; he can flex and extend the left forearm on the arm fairly well, but has slight difficulty in fully extending the right forearm. He is unable to raise either arm vertically from the side. He can raise the left arm to the horizontal position, but can only abduct the right arm about 6 inches from the side. He can place the left hand on the right shoulder, but can only with great difficulty place the right hand on the left shoulder. There is no loss of any sensibility in either upper extremity. No reflexes on striking the wrists or back of humerus.

The muscles of the right forearm and arm show slight tremors on movements; those of the left upper extremity do not.

The patient can execute all the movements of head and neck quite freely.

Electric Reactions of Muscles and Nerves of upper extremities:—

FARADIC CURRENT.

			RIGHT.		LEFT.
Flex. com. digit.	110 mm.	..	125 mm.
Dorsal inteross.	110 mm.	..	130 mm.
Biceps..	80 mm.	..	110 mm.
Triceps	70 mm.	..	100 mm.

GALVANIC REACTIONS.

			RIGHT.		LEFT.
Flexor subl. digit.	..	KCC	15c. 17°	..	10c. 7°
		ACC	10c. 18°	..	10c. 10°
		AOC	30c. 25°	..	30c. 24°
		AC Tet.	30c. 28°	..	35 28°
Ext. com. digit.	..	KCC	15c. 18°	..	15c. 18°
		KOC	45c. 30°	..	40c. 30°
		ACC	10c. 15°	..	15c. 18°
		AOC	35c. 30°	..	30c. 30°
		AC Tet	35c. 30°	..	40c. 30°

ONE ELECTRODE ON ERE'S POINT AND THE OTHER ON STERNUM.

Right arm	KCC	15c.	20°
			AOC	20c.	20°
			ACC	25c.	20°
Left arm	KCC	15c.	20°
			AOC (marked)	20c.	22°
			ACC (faint)	25c.	20°

DIRECT GALVANIZATION OF ULNAR NERVE.

RIGHT.	LEFT.
KCC 15c. 10°	10c. 10°
ACC 25c. 20°	20c. 15°
AOC 20c. 15°	15c. 15°

We notice therefore again marked degenerative reaction in the affected muscles.

The patient's condition altered very little for the next few months, and he left the Infirmary, Dec. 12th, 1883, but was re-admitted on Feb. 14th, 1884. It was found that the patient had now almost completely lost the power of his upper as well as his lower extremities. He now lies in bed perfectly helpless, is not able to raise himself in the least, and is unable to feed himself, or perform any other movements with his arms and hands. He is just able to flex and extend the fingers of both hands slightly; all the muscles of both upper extremities are markedly atrophied; there is, however, no "main en griffe." The muscles are flaccid. On striking the wrist, and also the back of arm, marked reflexes are seen.

Both lower extremities are completely paralysed. The muscles of the trunk also show now more atrophy and paralysis, especially the abdominal and pectorals. The muscles moving the neck and head, however, are normal. There is not any loss of sensibility in any portion of the body. Bladder and rectum act regularly. The tongue is clean, appetite good; there is slight abdominal distension. The movements of the chest walls are slight, the breathing is vesicular; there is no dyspnoea and no cough. Number of respirations 27 per minute. The heart's action is normal; the pulse 85, weak and compressible. The skin is moist, temp. 97°·5. The patient sleeps well, has no headache, and no cerebral symptoms. He remained in this condition up to June 15th, 1884, when it was noticed that the diaphragmatic and intercostal breathing became weaker, the respirations increased to 36 per minute; the patient's voice also became much weaker, and the expression of his face assumed an anxious appearance. Early on June 16th he had a severe epileptic convulsion, in which he died.

The post-mortem was made by Dr. Maguire, at the time

pathologist to the Manchester Infirmary. The thoracic and abdominal organs were found healthy, with the exception of the lungs, which were found œdematous and the seat of some emphysema. The brain and its membranes were normal; the brain on section showed many floodpoints; the sinuses did not contain an excessive amount of blood; the ventricles of the brain were not distended; cerebellum, pons and medulla appeared normal to the naked eye. The membrane of the spinal cord appeared healthy, and the cord itself was of a firm consistence throughout. Sections through the cord showed already to the naked eye changes in the anterior horns; these appeared depressed, highly pigmented, and in some few places minute spots of hæmorrhage were visible. The spinal roots and peripheric nerves appeared healthy. All voluntary muscles, with the exception of the muscle of the neck, had a pale appearance and were considerably atrophied. The spinal cord, posterior and anterior roots, a few spinal ganglia, many of the peripheric nerves and portions of muscles were kept for microscopic examination. Likewise the medulla, pons and portions of the cerebral cortex.

Microscopic Examination.—The muscles showed the usual changes; atrophy of the muscular fibres with loss of striation, multiplication of the muscle nuclei and cell-infiltration between the muscular fibres.

The small nerves in the muscles were found healthy.

The peripheric nerves, spinal roots and spinal ganglia showed no noteworthy changes. Some of the nerves, like the sciatic, seemed rather thinned; the several bundles of nerves were surrounded by fibrous tissue, containing a good many fat cells—and the sheath of the nerves showed fatty infiltration. The several nerves, however, showed normal relations, their axis-cylinders were well marked, and the medullary sheath appeared normal.

The spinal cord showed extensive and well-marked changes, chiefly in the anterior horns, and it may be said, without exaggeration, that throughout the greater length of the spinal cord, from the lumbar to the lower cervical region, not one normal ganglia cell could be found in the anterior horns.

In the lumbar region the affection was entirely confined to

the anterior horns; the ganglia cells were here found markedly atrophied, merely appearing as shining little specks; in the anterior grey matter there were also found granule cells, fatty detritus, and a few Deiter's cells were seen. The blood-vessels near the central canal showed a thickening of their inner and middle coat, and some peri-arterial cell-infiltration; small hæmorrhages were seen in the neighbourhood of the vessels.

The white matter and grey matter in the posterior horns showed increase of neuroglia, many Deiter's cells and blood-vessels, filled with blood-corpuscles and with their walls thickened.

In the dorsal region exactly the same changes were seen as in the lumbar region, with the addition of some sclerosis in the lateral columns, namely marked increase in the neuroglia, with atrophic changes in the nerve elements. The ganglia cells in Clarke's columns were less affected than the rest of the cells in the anterior horns. Sections from the dorsal region stained after Weigert, did not show the many small nerve-fibres seen in the healthy cord in this region. In the cervical region the changes were similar. In the lower part of the cervical region there was again lateral sclerosis. The changes in the ganglia cells in the anterior horns showed a less advanced condition of atrophy; some of the cells, though shrivelled, yet had a few atrophied outrunners; in some the nucleus even could be made out. The hæmorrhages near the central canal and the peri-vascular changes were better marked here than in the dorsal region. In the upper cervical region the lesion did not affect the lateral column, and was entirely confined again to the anterior horns. But here also the neuroglia throughout the white matter showed increase of Deiter's cells and peri-vascular cell-infiltration.

As the disease process in this case had been an ascending one, I requested Mr. A. Robinson, who is working in the pathological laboratory, to examine carefully the higher cervical region, medulla and pons, so as to study, if possible, the commencement of the pathological changes. Numerous sections were taken from the mid-cervical region upwards to the pons at the level of the root of the 6th cranial nerves,

and stained, some with carmine, some with osmic acid, and others with osmic acid and picrocarmine.

The changes Mr. Robinson describes as follows:—

The large ganglia cells of the anterior horns had almost entirely disappeared, a few only remaining at the anterior and inner portion of the anterior horns. These remaining cells were all more or less altered; some formed globular masses containing granules of a dark-brown substance, which completely shrouded the nucleus; others contained fewer of these dark-brown granules, evidently situated in the cell protoplasm between the nucleus and the cell wall. The processes of some of these latter cells were broken and irregular, and, after treatment with osmic acid, were found to contain masses of small black granules, while the processes of others were intact.

A few cells presented the appearance of a yellowish zone intervening between the nucleus and the cell wall; the whole cell having a glassy look. The neuroglia of the grey matter was very distinct. Deiter's cells were large and extremely numerous, but much smaller and finer in the grey matter than in the white columns to be afterwards described.

The blood-vessels in the grey matter were swollen and filled with red corpuscles. Their walls were thickened, the middle coat in many instances presenting the appearance of a clear hyaline band. And here and there in the anterior horns and grey commissure small hæmorrhages were found; two of somewhat larger size than the rest, just visible to the naked eye, were placed one on each side of the central canal.

The posterior horns presented no abnormal appearance.

White matter.—The neuroglia in the anterior and posterior white columns was normal; but in the lateral columns Deiter's cells were increased, both in number and size; their thick, anastomosing processes enclosed islets of nerve fibres, some of which were enlarged, and their axis cylinders were swollen. After treatment with osmic acid, dark granules were found external to the axis cylinders in the swollen fibres. The blood-vessels in the white columns were engorged, especially so in the lateral columns, where minute hæmorrhages were found between the nerve fibres. Ascending towards the decussation

of the pyramids, the nerve cells in the anterior horns became more numerous, and a few, at the inner and anterior portion of the anterior horns, appeared to be normal; but the majority presented some of the before-mentioned changes, and all stages were seen from the cells presenting only a yellow zone round the nucleus, and a generally glassy appearance, to the swollen globular cells, whose nuclei were hidden by a dense accumulation of dark-brown granules.

In the lateral columns the sclerotic changes became less and less marked, and could not be traced into the decussation.

The blood-vessels both of the grey and white matter were engorged, and small hæmorrhagic points were scattered in the grey and white substance, but specially in the former.

At the region of the decussation of the pyramids the morbid changes were less evident; but in the nucleus lateralis a few degenerated ganglion cells were found, while throughout the whole breadth of the cord the blood-vessels were engorged, and small hæmorrhagic points were scattered.

At a higher level (the commencement of the olivary nucleus), hæmorrhages, appearing to the naked eye as minute brown spots, were found in the central raphe, and smaller hæmorrhagic points were scattered irregularly over the section. The ganglion cells in the olivary nucleus, the nucleus gracilis and nucleus corneatus were intact, as were also the large cells of the hypoglossal nucleus; but the cells in the nucleus of the spinal accessory were loaded with dark-brown granules. No abnormal changes were visible in the remainder of the tissue of the medulla at this level.

Still higher, where the floor of the central canal opened out into the floor of the fourth ventricle, the same appearances were still presented; the nuclei of the hypoglossal nerves were healthy, but the cells in the vagal nuclei were loaded with dark-brown granules, and some of these cells had lost their processes and become globular. At this level, blood-vessels on one side of the medulla, running just below the nuclei of the vagus were greatly distended and engorged; at a slightly higher level this vessel was ruptured, and the effused blood had separated and destroyed the cells of the nucleus of the vagus on that side.

Above this level the abnormal appearances gradually ceased, and in the pons the nuclei of the 5th, 6th, and 7th nerves were healthy. The nucleus of the glossopharyngeal nerve was healthy, and in the pons the only abnormal appearance remaining was a dilatation of the small vessels and their engorgement with blood; but the hæmorrhages were no longer found.

The changes here described resemble very much those recorded by Cornil and Lépine,¹ especially as regards the vascular changes and hæmorrhages. From the fact, that these vascular changes were well marked in the highest cervical region, and also in the medulla, where as yet the changes in the ganglia cells were much less marked than in the lower cervical, dorsal, and lumbar regions—I am inclined to believe that the process is of a chronic inflammatory nature, with changes in the blood-vessels and hæmorrhage in their neighbourhood, and that the atrophy of the motor cells is secondary to the vascular lesions. That the vascular lesions often play the primary and most important part in many spinal diseases is seen from the recent observations on sclerosis in patches, diffuse spinal sclerosis, &c., where the vascular lesions are looked upon as the primary cause. The grey matter, being softer in structure and richer in blood supply, is even more likely to be affected by primary vascular changes, and the atrophy of the large ganglia cells would be easily explained.

There is only one point more in connection with this case to which I wish to draw attention. It was noticed during the latter part of the patient's illness that the so-called tendon-reflexes of the upper extremities were present, though there were no contractures, and it will be noticed that in the upper dorsal and lower and middle cervical region the spinal cord showed distinct lateral sclerosis. As the reflexes were noticed (though they had been looked for before) after the paralysis and atrophy of the limbs had set in, one cannot help thinking that they were secondary to the changes in the ganglia cells, and that this case, apart from the different clinical course, cannot be looked upon as a case of amyotrophic lateral sclerosis.

¹ 'Gaz. Méd. de Paris,' 1875, No. 11.

CASE IV.

The next case I have to report is one which shows the difficulty of forming an accurate diagnosis. The patient when admitted had paralysis and atrophy of the lower extremities, and also of the muscles of both hands and forearms, the extensors being chiefly affected; there were also some bladder troubles. These symptoms came on within a few days, and were accompanied by pains in the lower extremities; the tendon-reflexes in the lower extremities were absent, those in the upper extremities were present. Soon after admission the patient had complete anæsthesia of the lower extremities and abdomen and chest, up to the third rib on the left side and sixth rib on the right side. Three weeks after admission the patient had a slight attack of pneumonia, with albuminuria. Though there was only the history of gonorrhœa with orchitis, the patient was submitted to an energetic antisymphilitic treatment, and recovered completely in a comparatively short time. I happened to see the patient a few days ago, and he was then in perfect health, being able to walk twelve to fifteen miles with ease. There is now no trace of either paralysis or atrophy, and the tendon-reflexes of the lower extremities have reappeared.

The following is a brief outline of the history of the patient's illness.

Alfred K., fishmonger, æt. 45, was admitted into the Infirmary on Feb. 18, 1884.

The patient has lived in Manchester for the last fourteen months. Seven years ago he came to England from India, having been there twelve years in a regiment of Lancers. During his stay in India he had a bad attack of dysentery in 1871, but he never had ague; he has not had syphilis, but twenty-seven years ago had an attack of gonorrhœa, followed by the swelling of one testicle some months after; since then he never suffered from rash, sore throat, or any symptoms pointing to syphilis. He has been rather intemperate, and taken alcohol freely and regularly for twenty-five years. He has been married for twenty-four years; his wife had one miscarriage twenty-three years ago; after this she has had two children, of whom one died when six years old from some brain disease.

The patient's father died at the age of 84, his mother at 80. He has a brother and sister living who are healthy; two brothers and one sister died; the nature of their diseases he does not know.

Since his return from India the patient's health has been exceptionally good, and he has not suffered in any way.

Ten days before admission he noticed his legs giving way; his walking rapidly became worse, so that six days before admission, therefore in the course of four days, he was unable to walk at all; four days ago he noticed his hands becoming affected, and he was unable to hold anything in them. He was troubled at the same time with pain in his legs, and a distinct girdle pain round the abdomen; he was unable to control his bladder, and his bowels were confined; and after taking aperients he noticed that he had lost also some control over his sphincter.

Condition on Admission.—The patient lies in the recumbent posture, from which he can only rise with great difficulty; he is not able either to stand or walk, nor can he grasp or hold anything in his hands. There is marked atrophy of the lower extremities, and also of the hands and forearms; there is some slight œdema over dorsum of foot and lower end of leg on each side. There are several recent bruises on both legs, which the patient says were produced by repeated falls at the commencement of his illness. There are no enlarged glands to be felt anywhere, and the appearance of the face is healthy, and contrasts with the condition of the limbs. There is marked arcus senilis. There are no cerebral symptoms; the special sense-organs and cranial nerves are normal; the muscles of face and neck are unaffected.

Upper Extremities.—On the right side the shoulder and arm-muscles show considerable amount of power; the extensors of the wrist and finger are very weak; the flexors much less so. He can supinate and pronate, though to passive resistance these muscles appear weak; he cannot flex the index-finger or thumb, and is able to adduct the thumb and oppose it to the hand but slightly. There is marked atrophy of the thenar and hypothenar eminence, and of the muscles of forearm. The patient complains of pain in the shoulder, arms and hands.

No tendon-reflexes can be elicited. The left upper extremity is affected in a similar way; only to a still greater extent, the patient is unable to raise his left arm above his head; resistance to passive movements is weaker, and the paralysis both of the extensors of the wrist and of the fingers more marked. Tendon-reflexes are present on the left wrist and above elbow.

Lower Extremities.—The patient is unable to extend the foot or any of the toes on either side; he is unable to extend the knees or thighs, nor is he able to adduct or abduct either leg; he is, however, able to flex the knees and also the thighs, though there is not much power in the flexors, as seen by their resistance to passive movements. There is marked atrophy of both legs and thighs, and the muscles feel flabby. On the whole the left lower extremity is weaker than the right. The superficial and deep reflexes are absent. The patient complains of pain in his legs and thighs. Some of the paralysed muscles show fibrillar twitchings. The cremaster, abdominal and epigastric reflexes are absent. The presence of scapular reflex is doubtful. The patient has some loss of control over his bladder, and also over his rectum.

The examination of the thoracic and abdominal organs shows nothing abnormal. The urine has sp. gr. 1018, is faintly acid, contains neither sugar nor albumen.

Feb. 21.—The patient complains of numbness of his legs, and examination shows an almost complete anæsthesia of both lower extremities, and extending on the left side to the crest of ilium, both front and behind, and on the right side as far as umbilicus.

Feb. 22.—The anæsthesia remains stationary on the left side; on the right side it now reaches a line corresponding to lowest point of ensiform cartilage and passing in a semicircle right round the back to the middle line. Above the line of anæsthesia there is a small band of hyperæsthesia. There is no anæsthesia of the upper extremities. Sense for pain and for temperature appears normal in the anæsthetic region.

Feb. 23.—The anæsthesia has now reached on the right side a line corresponding to the fifth rib, and passing round to the middle line in the back. The anæsthesia on the left side has increased a little. The condition of the patient has not undergone any other changes.

The patient is now ordered mercurial unction and iodide of potassium, twenty grains per dose, three times daily; also a chlorate of potash gargle.

Feb. 24.—The upper limit of the anæsthesia on the right side has now reached the third rib. The condition is otherwise unchanged.

Feb. 28.—The anæsthesia is rather less; the patient noticed some slight improvement of his paralysis. He can move the toes of his right foot slightly and can extend the right wrist better. The left upper and lower extremity remain in the same condition.

From this date the patient's symptoms began to improve.

The electric reactions taken at different time showed degenerative reaction, as shown from the following data, observed on February 28th :—

FARADIC CONTRACTILITY OF MUSCLES.

Upper Extremity :		RIGHT.		LEFT.
Biceps	80 mm.	..	76 mm.	
Triceps	70 mm.	..	65 mm.	
Extensores digit. ..	20 mm.	..	15 mm.	
Flexors of digits ..	40 mm.	..	39 mm.	
Lower Extremity :				
Tibial antic. ..	60 mm.	..	60 mm.	
Extensor digit. long.	35 mm.	..	30 mm.	
Quadriceps	70 mm.	..	65 mm.	

GALVANIC REACTIONS, DIRECT MUSCULAR.

Upper Extremity :		RIGHT.		LEFT.
Extens. digit.	KCC - 15c.	25		
	ACC - 10c.	15		
Flexor digit.	KCC - 15c.	20c.		
	ACC - 15c.	15c.		
Lower Extremity :				
Tibial antic.	KCC 10	15		
	ACC 10	10		
	(strong contr.)			
Extens. long. digit. ..	KCC 15	15		
	ACC 15	10		
	(strong.)			

The other muscles were tried on other days with similar results.

On March 7th the patient's temperature, which up to now had always been nearly normal, suddenly rose to 100, and he

complained of pain in his right chest. Physical examination on March 8th showed dulness over the right base behind, and crepitation with every inspiration over the dull area.

On March 9th the temperature was $101^{\circ} 2$; the physical condition of the chest the same as before. The patient brought up some rusty sputum. The paralytic symptoms had remained stationary the last few days. The urine contains a small trace of albumen, sp. gr. 1023,—scanty.

After this attack of pneumonia the patient's condition rapidly improved. The lung symptoms had disappeared by March 23rd, the albumen a few days previously.

The anæsthesia had considerably receded, and was now (March 23rd) limited to the legs and thighs, but extending higher on the right than on the left side. The patient is able to extend his toes slightly; he can also flex the index-finger and thumb of right hand and slightly extend the right wrist. The left upper extremity is recovering much slower. Several of the muscles of the lower extremity, such as the vasti interni, the adductors of the thigh, and the quadriceps, act much better.

By May 31st, 1884, the patient had sufficiently gained power to be able to walk with the help of sticks, still dragging the legs, owing to the weakness of the extensors. The arms and hands have improved most markedly, and he can now grasp fairly well and perform even delicate movements with his fingers. The anæsthesia has completely disappeared. The superficial reflexes are now present, but the deep reflexes have disappeared. There are still the tendon-reflexes in the left upper extremity, but much less marked.

The iodide of potassium had been continued, and up to the present (May 31st), the patient had forty inunctions. For the last month he had been treated with weak galvanic currents.

On July 14th the patient was fit to be discharged, and he was sent to the Convalescent Hospital at Cheadle, where he remained for two months. He returned completely cured, and is now perfectly well. The patellar tendon-reflexes, which had remained absent for some time yet, have now reappeared.

As regards the nature of the lesion in this case, I am inclined to believe that it was diffuse central myelitis,

with some meningitis, probably of syphilitic nature. The symptoms correspond closely with those seen in two cases described by Déjérine,¹ and in a case given by Putnam.² These cases terminated fatally, and the lesion was chiefly an inflammation affecting the anterior and posterior grey matter. The anæsthesia being so distinctly limited, and being more marked on the right side, while the paralysis and atrophy were more pronounced on the left side (especially in the arm and hand), one might think of a syphilitic tumour; yet if this had been the case, and if the tumour were situated in the cervical region, we should have expected spastic paralysis of the lower extremities, and not atrophic paralysis. The continuity of the motor and sensory troubles from the lowest lumbar to the cervical region, strongly support the view, that a diffuse central myelitis affecting the grey matter was the cause of the symptoms observed. The implication of the bladder and rectum, apart from the exact linear limitation of the anæsthesia, show at once that the case was not one of peripheric multiple neuritis, or alcoholic paralysis.

¹ 'Revue de Médecine,' Jan. 1884.

² 'Journal of Nervous and Mental Diseases,' 1885.

(To be continued.)

AN EXPERIMENTAL ENQUIRY INTO THE NATURE OF THE OBJECTIVE CAUSE OF SENSATION.

BY JOHN B. HAYCRAFT, M.B., B.SC., F.R.S.E.

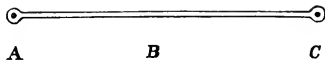
Professor of Physiology, Mason College, Birmingham.

THE present lecture, recently read before the Birmingham Philosophical Society, is an extension of a short communication to the Royal Society of Edinburgh,¹ and includes an account of some experiments conducted in my laboratory in the Mason College, together with a discussion of some general psychological problems.

If a stimulus, say a rapidly interrupted electrical current, be applied to the body for a definite period of time, it will of course produce a sensation; but that sensation will not coincide in time with the stimulus that produces it. In the first place, the sensation will not commence as soon as the stimulus is applied, and secondly, it will continue after the withdrawal of the stimulus. In the first part of this paper we shall discuss this non-coincidence in point of time between duration of the sensation and the stimulus.

The sensations have their seat in the brain, and the impressions require a definite time before they can reach this part of the body, situated as it is in so central a position.

FIG. 1.



Let A, Fig. 1, represent a particle (cell) at the surface of the body which is stimulated; B, a nerve passing from it towards a sensitive particle (cell) C in the brain. A definite time

¹ Proceedings R.S.E., 1883, p. 246. Abstracted in "BRAIN," April 1884, p. 141.

must elapse before A is excited by a stimulus, and the impulse thus set up has time to travel along the nerve B and throw into excitement the cell C. A nervous impulse travels no faster than some forty yards in a second, and from this factor alone—allowing three feet for the average length of a nerve path—the sensation will be delayed one-fortieth of a second.

But the sensation remains after the stimulus has been withdrawn. If I pin a small patch of red paper near the edge of this wheel, and then cause it to revolve, you will observe that the red patch moves round, then it appears to elongate, and finally, as the speed of rotation increases to some ten or fifteen revolutions per second, it is seen in the form of a complete red ring. You will remember, too, that in the firework known as the “Catherine Wheel,” one part alone is alight, yet, when it revolves, one sees a ring of flame.

What is the explanation of this? At any given instant the patch of red or the coloured flame is at one spot only; how is it seen as if it were at all points of the circle in which it moves? This is very easily explained, for the sensation produced by the patch remains after the latter has moved away, and if we suppose that the patch starts from a certain point and revolves so rapidly that it comes back to that same point in a period of time less than the time that the sensation remains, we shall be sensible of a continuous ring.

This may be demonstrated in another and perhaps a more conclusive way. I will attach the patch of red to a steel spring, which can be caused to vibrate. I will now set the spring in such a manner that it will appear five times during each second above this screen, disappearing five times below and behind it. You are now conscious of a flickering sensation, but on altering the spring, so that it may vibrate ten times instead of five, the red patch appears continuously in front of the eye. The red object is, however, being continually withdrawn from view for periods of one-twentieth of a second. The sensation must, therefore, persist for this time.

In the case of hearing, Helmholtz found that when the “beats” follow one another at the rate of twenty or thirty a second, they cease to produce individual impressions, fusing

into a jarring grating, rough sound, which may be heard even when 130 beats affect the ear during the same interval.¹

Physiologists tell us, that in the case of tactile sensibility no less than 1,400 impressions must stimulate the finger tip before they become fused. This statement is or is not correct, according to our interpretation of the word "fused." If it is meant that then, for the first time, they cease to produce individually a sensation, it certainly is not true. At most it is a very superficial description of what may with care be observed. Touch this revolving toothed wheel. On turning it very slowly, each little tooth will be felt in its turn, and will be recognised by a separate feeling of impact.

Increase somewhat the speed of rotation and it will be no longer possible to recognise each separate impact, but a sensation will be produced which we call "roughness." Rotate more rapidly, and the wheel will feel of a finer and finer roughness, until, when 1,400 teeth are brought in contact with the finger tip during the period of a second, it will feel like a rotating smooth wheel producing merely a shearing or displacement of a portion of the skin touched.

There are, then, *three* periods. In the first, we can distinguish in consciousness each individual impact and the gap which separates it from the next impression. In the second, they have lost their individual character and produce a sensation—"roughness." In the third this feeling changes into that of smoothness.

These periods are not sharply demarcated one from another, yet one may state approximately in one's own experience where one begins and the other ends. In my own case impacts as rapid as forty per second are recognised as distinct; forty to 1,500 produce roughness, and above that number a sensation results similar to that produced by a rotating smooth wheel. The number forty is probably too high. With very strained attention, I sometimes fancy I can distinguish as many impacts in a second. Perhaps it would be safer to put it down at thirty.

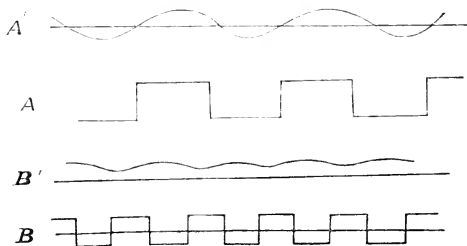
These numbers were determined by allowing a vibrating

¹ "The Sense of Tone, as a Physical Basis for the Theory of Music." Eng. Trans., p. 253. In English works Helmholtz has been generally misquoted. He never says that 130 beats may be heard individually in a second.

steel rod to strike the finger tip. Its pitch could be varied at will by weighting it or by altering its length, and could exactly be determined by arranging it so as to write on a smoked cylinder revolving at a known speed.

We can see, from the results of this experiment, striking analogies between the sense of touch and the senses of sight and hearing. In the case of the red object attached to the steel spring, in the case of the beats, which are intermittent sounds, and in the case of the revolving toothed wheel, we find that on increasing the frequency of the stimulus the sensations pass through three distinct stages. In each case the slowly intermitting stimuli are formulated separately in consciousness, each is followed by a period of rest, and then another is felt. Such slow periodic stimuli may be productive of great pleasure.

FIG. 2.



Slow beats may produce considerable artistic effect; the ticking of a watch and the "passes" of a hand over the skin may induce hypnotism. Slow intermittent stimuli are pleasurable and soothing—instance the rhythm of poetry and the rocking of a cradle.

Let the line A indicate the time of application of a slowly intermitting stimulus. The line A' shows the duration of the resulting sensation. The two lines are arranged in vertical series. When they rise above the abscissal line, they indicate respectively that at that point the stimulus is being applied or the sensation felt. On striking below the abscissal line, the stimulus is being withdrawn in A, and the sensation ceases to

be felt in A'. These lines indicate in a graphic way the condition of things in this first stage; two sensations being separated one from another by a period of rest—the curve descends below the abscissal line.

When, however, the frequency of the stimulation is increased, so that they are no longer individually felt, you obtain a continuous sensation, which varies periodically at first in its intensity, and indeed in the first part of this stage we may be conscious of this variation. This may be graphically represented (B and B'). The curve B represents the stimuli which are seen to follow one another more closely. In B' the sensations have not time to disappear before the result of the next stimulus follows on.

The sensations are now as unpleasant as those produced by slow intermission are pleasant in their nature. Few things are more annoying than the flickering of a gas jet. Helmholtz has shown, that the cause of dissonance in music occurring when certain notes are sounded together is caused by the *rapid* beats they produce. When this wheel revolves so that thirty or forty teeth touch the finger tip, you remark that it feels rough, a term standing for a sensation or idea which is not of a pleasant kind. Thus we say "a rough fellow," meaning thereby a man whose activities are not of an agreeable nature. In the case of touch, and also of hearing, this second stage is much prolonged, being still of a disagreeable nature, although the sensation is absolutely continuous.

In the third stage the sensation is also continuous, and is not to be distinguished from a stimulus constantly applied.

Why stimuli, differing only in their frequency of application, should produce such different effects on the organism we cannot say. This question is, to a certain extent, out of the range of present experimental inquiry, and its consideration must therefore be postponed. It is enough that we can state this general psychological law, which applies to senses such as touch, sight, and hearing, where the sensations coincide pretty exactly in time with the stimuli which produce them, but not for the senses of temperature, taste, and smell, where the limits of the sensations are very diffuse. "Slowly intermitting stimuli are followed by slowly intermittent

sensations, which intermittence is pleasurable, provided the stimulus itself be not of a disagreeable nature. Increase the frequency, and the sensations become fused into a disagreeable rough sensation; and finally, in the third stage, they produce a continuous smooth sensation, not to be distinguished in quality from the same stimulus constantly applied." It is a curious fact, that in tactile sensibility the same sensation is produced, whether a single portion of the skin be stimulated in rapid succession, or a stimulus be rapidly applied to a series of sensory surfaces, one after the other. Roughness is produced, whether you place your finger against a revolving wheel, or draw the smooth edge of a knife, held vertically, along the front of the finger. In all cases it is combined with tickling, if the stimulation be very light. Hold a tuning-fork, set in vibration, against the lip; it tickles intolerably; draw a pin along the lip, and the same feeling is produced. Of course you are aware of the difference between the two, because the location is different. Both produce tickling. In one case this is associated with contact at one spot, in the other with contact shifting along the lip. The feeling of tickling and of roughness seems, then, to be produced whenever a series of nerve impulses, following one another in rapid succession, reach the sensorium, no matter whether they all came from one sensory point, or each one from a different sensory surface.

As no experiments, so far as I am aware, have been conducted upon the coincidence in time between the stimuli and sensations in the sense of temperature, I undertook a series, a portion of which I will now describe.

Touch this iron ball, which is a good conductor of heat, and you will be conscious at once of a sense of impact, but only after a very appreciable interval will you feel that it is cold. On withdrawing the hand the sensation of cold remains for some time. Obviously, the coincidence in time is not so exact in the sense of temperature as in touch. For more exact study of these phenomena I have used a very simple piece of apparatus. A long and slowly vibrating rod, 3ft. to 4ft. long, was fixed to an iron vice, and to its free end a glass bottle was attached. To this was fitted a cork, through which passed an

iron rod. The bottle was filled with a mixture of ice and salt, which cooled the rod in its whole length. The vibrating rod brought the cooled rod periodically upon the finger tip; it was held there for half a second, or long enough for it to be felt as a cold body, and then allowed to swing back. It was found that if the iron was brought periodically in contact with the finger, each time after the lapse of a second a continuous and uniform sensation of cold was produced; if after a second and a-half, each impact was associated with a *separate* sensation of cold. The sensation remains for more than a second after withdrawal of the stimulus, but not for so long as a second and a-half. As will be presently shown, the same results are not obtained when other parts of the body are stimulated.

Sensations of cold and heat, as well as of impact, result from stimuli applied to the surface of the skin. Remove this, and the subjacent bones and muscles are found insensible to these stimuli. Now the skin is a large and extensive tissue, and microscopical sections taken from various parts show considerable differences. Some parts are more richly supplied with nerves than others, and these are regions where sensation is acute. In many places the horny outer layer, into which the nerves do not pass, is very thick, and it may be developed into protecting structures, such as nails and hairs. The experiments on tactile and thermal sensibilities, recorded in this paper, have been made with the skin of the finger tip. Let us extend the enquiry, and examine other parts of the skin. It is well known that all parts of this tissue are not equally sensitive to touch. The skin of the finger tip and the front of the hand can be stimulated by the impact of a lighter body than will affect the skin of the back of the hand. Again, the skin of the front of the arm is more sensitive than that of the dorsal surface, and still more so than the skin covering the shoulders. Together with this difference in actual sensibility, the brain is unable to localise impacts which affect the less sensitive parts so exactly as those which affect the more sensitive parts. If the points of a pair of compasses, less than one millimetre apart, touch the finger tip, they may be distinguished one from the other, but over the skin of the

back they may be held over an inch apart, and yet give rise to only one impression. This difference, not to be discussed here, is due (partially, no doubt) to the arrangements of the peripheral nerves. What I propose to discuss is, whether the non-coincidence in time between the stimulus and the sensations varies with the sensibility and powers of localisation of the surface.

If a rod, vibrating some twenty times a second, be held to the finger tip or lips, sensations of distinct impacts may be felt. Allow the rod to vibrate upon a surface less sensitive, say the skin over the sternum, and the impacts are individualised almost as before. I should mention that inasmuch as the skin is less sensitive, the amplitude of the vibration and force of the impacts must be increased. There is little difference then, in this respect between the more sensitive and less sensitive parts. On experimenting with the toothed wheel applied to the sternum, it is impossible to obtain the finely rough sensations with rapid rotation which could be felt by the finger tip. This is probably due to the less sensitive nature of the surface, and the impossibility of pressing the wheel upon the skin in order to compensate for this without producing at the same time injury to the surface. We see, then, that in less sensitive parts the sensation coincides in time almost as exactly with an applied stimulus as in the case of the more sensitive parts, whereas the space localisations are so different.

Let us now extend our observations on the sense of temperature to other parts of the body. Apply after intervals of a second a good conductor—say an iron ball—to the skin of the bare arm; distinct sensations will be recognised on each application, whereas a continuous and unbroken sensation is produced on touching the ball in the same manner with the finger tip. This is not due to the fact that the arm is usually protected, and in consequence might be conceived as less inured to cold, for the same obtains with the skin covering the forehead and lips. The difference is partly due to variations in the thickness of the epidermis—a bad conductor—which separates the body touched from the nerves situated in the lower layers of the skin. The corneous fingers of a working-

man allow but slow conduction and a sensation is perceived only after a lapse of two or perhaps three seconds.

In our study of the senses of hearing, sight, and touch, I have shown that we are able to perceive twenty or thirty sensations in a second, and, but one, or at most two, sensations of cold or heat may be felt. We find that the sensations of taste and smell, like those of temperature, spread over a longer period of time, and are not sharply limited in duration.

Herbert Spencer, in his "Principles of Psychology" (vol. i., p. 169), calls attention to this difference, distinguishing between peripherally initiated feelings caused by internal disturbances—some of which he says are extremely indefinite, and few or none definite in a high degree—and feelings caused by external disturbances which are mostly related quite closely alike by co-existence and sequence. He illustrates this by the fact, that our states of consciousness in connection with vision and hearing are more sharply limited in time and space than those in connection with smell and taste, and, still more, hunger.

Now, discarding the fact that when considered developmentally, the retina at any rate is far more internal than the mouth or nose, the former being really a portion of the brain, the latter puckerings in of the surface, I would suggest that the all-important factor producing this difference is not anything in the brain, nor in the nature of the feelings themselves, as Herbert Spencer seems to me to indicate, but in the nature and arrangements of the end organs. If the point of a pin impinges upon the finger tip, the epithelium is depressed, and at the same time the nerves of tactile sensibility are stimulated, and on withdrawing the pin they are in a condition of rest. Also in the ear, the sound vibrations travelling rapidly into the internal ear cause the structures there (the basilar membrane and rods of Corti) to vibrate, and—as we are aware from our knowledge of the action of one vibrating body upon another—they do this very rapidly.

Far otherwise with the senses of smell and taste. When odorous particles pass into the olfactory or upper part of the nasal cavity—in which the nerves of smell are placed—it is by diffusion from the lower or respiratory part of the nose. If

the breath be held, and a piece of incense paper be burnt in front of the face, some time may elapse before the scent is perceived, because the odorous particles have in fact to diffuse into a closed sac, slowly increasing in numbers. If, on the other hand, the experimenter "sniffs" the air, the stimulus will be more rapidly perceived, because a large number of odorous particles are carried rapidly through the lower chamber of the nose, inducing out-currents from the upper chamber, which consequently becomes immediately filled by these. In like manner, the closed sac has to get rid of odorous particles from within it by this same slow process of diffusion, before the odour ceases to be felt. The application and removal of the stimulus is then a gradual process. The feeling produced is therefore gradual in coming on and slowly passes off.

In the case of the sense of taste, the same holds true. Substances held in solution are alone tasted; the tongue is covered with a layer of mucin derived from mucous and salivary glands, and the nerves are not superficial but embedded in the epithelial covering. It will be easily understood that in this case also the accession of a sensation must, from the nature of the stimulus, and the position of the end-organ, be gradual in its production and slow in passing off, and therefore not strictly limited in time. The watery solution has to mix in the first place with the mucin covering the tongue, before it can reach the end-organs situated in the epithelium.

In the case of hunger again, the limitations in time are due entirely to a condition of things other than mental. The fulness of the alimentary canal is associated with a feeling of comfort, and when no food is present there with a feeling of hunger; and as there is every conceivable transition between a condition of full stomach and an empty one, so the passage of the one sensation into the other must pass through innumerable transition states.

If it be needed, another example may be mentioned in the case of the cold body applied to the horny tip of the finger, and to the thin skin covering the sides. The nerve-endings are stimulated by the addition or withdrawal of heat from the

nerves of the skin. This is gradual, and does not correspond to the application of the cold body to the surface because of the horny epithelial covering. Where this covering is thin, the limitation in time of the sensation is more definite than where it is thick; and could we apply the cold body directly to the nerve end-organ, the limits would then be very sharply defined. Cover the hand with a glove, and the limits would be very ill-defined indeed.

Sufficient evidence has, I think, been adduced to show that where the limits of a sensation are not well defined in time, we may conclude that this is not due to anything in the nature of the sensorium, but depends upon the way the external energy is changed into nerve energy in the terminal end-organ.

Let me define a sensation as the result of a transformation of the energy travelling along a nerve of sensation from without, into the energy manifested by the nerve cell to which it passes. We have, I insist, no reason to doubt, that if that nerve energy travelled twenty times a second along a nerve of smell, of taste, hearing, or of sight, we should be conscious of twenty separate sensations in each case.

That such a transmission is impossible in every case we know, but it is due to the fact, that in these cases the nerve cannot be stimulated from without so frequently.

A theory advanced, that quality in sensation depends for its objective cause upon the "frequency" of the vibration of the stimulus which produces it.

The air may be caused to vibrate at infinitely long or infinitely short periods, and at all intervening rates. If I pull this long steel band which is fixed to an iron vice and then let it go, it will vibrate and set the air in motion. It is now vibrating some ten times a second, but, although the amplitude is very great, you hear no sound. Shorten the band, increasing therefore its frequency of vibration, and you hear a low deeply-pitched note. Continue to shorten, and the note becomes higher, and higher, and at last you can hear nothing at all. We see that here, at any rate, the different sensations you were conscious of depended upon the frequency of vibra-

tion of the air set in motion by the vibrating steel band. The steel band—and the same applies to other sounding bodies—not only vibrates as a whole, but also in parts, so that the air is moving in rather a complex manner. We have the movement of the air corresponding to the movement of the band, as a whole producing what is called the fundamental tone; and in addition, movements corresponding to vibrations of parts of the band, and producing the “harmonic tones.”

Now, the sensory quality which distinguishes one instrument from another—the difference say in the “sound” of a string stretched upon the sounding-box of a guitar, and another attached to that of a violin—depends upon a difference which these boxes produce in the number and strength of these harmonics; upon, in fact, the intensification of certain frequencies. The multitudinous sensations of the Concert Hall or Opera are produced by immense numbers of little waves, varying infinitely in periodicity, falling upon and stimulating the ear.

Much the same may be said of sight. A something called “Ether” may be thrown into vibrations, and these cause the sensations light and colour when they fall upon the retina. When a ray of light is passed through a prism, the vibrations of which it is composed are separated out in a scale called the spectrum. At one end, in the region of the ultra-red, we have slow vibrations. Like the very slow vibrations of the steel band, they produce no sensation. Higher in the scale where the rapidity is greater (some 481 billions per second) a sensation called red is produced. As we pass higher in the scale, the sensation changes into orange, then into yellow, green, blue, and last of all violet. Where still more rapid vibrations are found, namely in the region ultra-violet, no sensation—again the analogy with the steel band is seen—is produced. We see here again, that it is frequency or rapidity of vibration which determines the quality of the produced sensation.

For the last few years I have held as probable, that much the same holds for the senses of taste and smell, believing that just as a certain vibration will produce, say, a sensation of colour or of sound, so the periodicity of the vibrating particle given off from the scented rose, or of the molecule in solution

will determine the sensation of smell or taste produced. This is now removed from the field of mere speculation, and one may look forward to a time, perhaps not far distant, when in a more advanced state of molecular physics one may be able actually to determine the rate of vibration of even complex substances; a time when the arrangement of a bouquet or menu may be as scientific as thorough-bass.

Professor William Ramsay, in a very valuable communication to "Nature" (June 22nd, 1882), has brought forward, I think, some very important facts as to the objective cause of smell. He finds that gases of very small molecular weight, and therefore of high pitch, are incapable, like the ultra-violet rays and the very rapid vibrations of the steel band, of producing a sensation. All the simple gases, and many of the compound ones, are odourless; in fact, a gas must have a molecular weight fifteen times as great as that of hydrogen, in order that it may be smelt. He has studied certain chemical series, for example hydrocarbons, such as the paraffins and olefines, which increase regularly in molecular weight. The lightest have no smell, but passing up the series odours may be perceived, which vary in quality, and increase as a rule in strength. There are many other points in this paper which show analogies between smell on the one hand, and sight and hearing on the other. It seems difficult, however, to find examples of very heavy gases which, like the ultra-red vibrations, produce no sensation at all.

I have myself been working at the sense of taste, and have been already able to arrange many familiar tastes in a scale or series, associating them in each case with definite molecular vibrations. The subject is not, however, so complete as I should wish, and I have postponed its publication.

I have been also able to arrive at similar conclusions in respect to touch. Let me ask you, Mr. President, once more to touch this revolving toothed wheel. I turn it very slowly, and you feel each separate tooth. Increase its rapidity they fuse into a sense of "roughness," and as I continue to increase the speed you pass through every possible degree of sensation from a coarse to a fine roughness, and finally, it is rough no more. We have here, in fact, quality of sensation depending

upon frequency of impact. There are a hundred grades of sensation intermediate between a very coarse and a very fine roughness. These are not grades of quantity, but of quality.

It is true, certainly, that there may be a difference in quantity, but that is because on rapidly rotating the wheel we cannot *press* the finger against it without fear of injury. It is equally true with many sound instruments. On increasing the revolutions of a syren the music is at first low and voluminous, then it becomes higher and less voluminous, but more intense. The higher notes of a piano are weaker than the lower ones.

A moment's consideration will suffice to show, that this "roughness" is as much a sensation, with qualitative variations, as taste and smell. It is incorrect to say that it is a sensation *of* intermittent stimulation, although it is a sensation *produced by* intermittent stimulation, which is quite another thing. From our very earliest infancy the objective cause of the sensation has been known to us, yet at first it must have been gained entirely by experience, which without the assistance of the eye would have been very difficult. We can no more be said to be conscious of 1000 impacts in a second of a toothed wheel, than of 256 vibrations of a wire when the lower C on the piano is sounded. In both cases we have definite sensations, and in both cases we have been able to associate these with their objective causes. A practised musician on hearing a note sounded might name it, and tell the period of the string that produced it. After long experience with the sense of touch, after careful attention to the sensations, after careful experiment conducted to find the exact frequency of impact producing these, it would also be possible to say of a given sensation, that it is produced by so many impacts per second.

It may further be objected, that an intermittent impact is not the same as a vibrating stimulus. In one case the skin is stimulated for a certain time, and then it is stimulated no longer, whereas when a sonorous vibration stimulates the organ of Corti it is thrown into periodic movement, alternating in direction, and without a break in its continuity. This difference is more apparent than real. A child upon a swing may be kept in motion in either of two ways. In the first case,

by a force applied, say by means of the hand, which shall adapt itself in every way to the pendulum action of the swing, varying in the direction of its application and increasing in amount as the lowest point is reached. This represents one vibration producing another. The vibrating arm causes the swing to vibrate. Or secondly, the swing might be set or kept in motion by short pushes, synchronous with the period of the swing. This, of course, represents an intermittent force producing a vibration. We see, then, that although the toothed wheel is applied periodically, and is not in itself a vibration, it may produce a vibration of the skin. Indeed, as the skin has "mass," this must be the case. In all cases, it is the vibration of the sensory surface which is transformed into nerve impulse, it matters not how this is produced. It need only finally be added that it is possible to stimulate the skin with true mechanical vibrations, which produce the same effect on the nervous system as the toothed wheel. Instead of the ordinary toothed wheel, use a grooved wheel, the border being regularly curved. Apply the finger to its border, or place a vertical metal spring, fixed at one end to the vice, between it and the finger, so that on revolving it pushes the spring alternately forwards and backwards. In this case the motion will be a true vibration. We have then in touch, as in sight, hearing, taste, and smell, not only quantitative variations, but in addition a *scale*, each part of which is quantitatively different from the rest.

There is yet another sense, that of temperature. Its study in this connection is most interesting.

Lord Bacon anticipated some of the results of the Dynamical Theory when he stated his belief, that heat was a kind of motion or agitation of the particles of matter. The ultimate particles of gases, liquids, and solids are probably in constant periodic movement, and these movements may be transformed into nerve impulses. As in the other senses, certain specific structures are necessary for this transformation; they are found in the skin. Bring a live coal near the hand; its particles are in a state of great agitation; it induces similar motions in the skin, and these are transformed into nerve impulses, which, passing to the brain, produce a sensation—

“heat.” If we stop to consider for a moment the various heat sensations of which we are conscious, we are struck with this very curious fact, that there is an entire absence of *quality* in this sensation. We may have in the other senses different qualities and kinds of sensation, and these may, in addition, vary in amount or quantity. There is nothing in the sense of temperature which corresponds with pitch in music, or colour in sight. We have “more heat” or “less heat,” but it is always of the same kind. We cannot distinguish the heat of a hot potato from that of a piece of iron at the same temperature.

We must not be deceived by the terms “hot” and “cold.” These are not qualities, but two quantities relative to the body temperature. In the same way we have light and darkness, sound and silence. Just as darkness may be less light, so cold is less heat—indeed the darkness, by common experience, may after a time become relatively light, and the cold may become relatively warm when afterwards compared with super-added sensations. Pass from a light into a dark room; you can see nothing. In a few minutes you become sensible of light. Plunge your hand into tepid water after warming it in front of the fire, and it feels cold. Now place the hand in ice-cold water, and then into the tepid fluid, and the latter feels quite warm.

There is, then, nothing of the nature of a qualitative difference in our sense of temperature, and curiously enough there is nothing which, according to our theory for the production of quality in other senses, would produce quality in this.

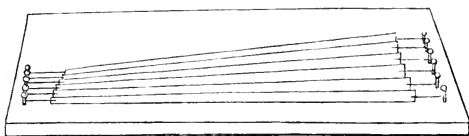
Physicists tell us, that when a body gains in temperature its molecules continue to vibrate at the *same rate or frequency*, increasing only in *amplitude*. When the skin is heated, from whatever source, its molecules do not vibrate either more quickly or more slowly than before. We have, then, objectively no difference in frequency of vibration, and subjectively no difference in quality.

The experiments of Kirchhoff and Balfour Stewart tend to show, that at very high temperatures certain “partial” vibrations become more pronounced, but before these temperatures

could be reached the skin would be entirely charred. These temperatures are therefore outside the range of our enquiry.

Returning once more to the study of tactile sensibility, the usefulness of a sensation will depend upon two things. In the first place, it will depend on the extent of the scale of quality which is present, and in the second upon the smallness of the difference of quality that we are able to appreciate. In sound we have some eight or nine octaves, which can produce sensations; and towards the middle of the scale a practised musician is said to be able to distinguish two notes one-sixtieth of a tone apart. In sight we have barely an octave, yet within this small scale we are very sensitive to minute intervals. How about the sense of touch? Taking twenty-five for the lowest number in the sense of roughness, and 1,500 as the highest, we have nearly six octaves; more than in sight, but not so many as in sound.

FIG. 3.



Within this scale what differences can we discern? To investigate this question I have constructed the following instrument, which may be called the "touch-board."

It consists of a series of steel bands radiating from a centre, and bounded by two concentric arcs. These are fastened by their edges to a board, and are kept "taut" by wires passing to metal pins. On the right-hand side the interval between the bands is one centimetre, but to the left it is only half a centimetre. On drawing the forefingers of the two hands across the bands with the same velocity and placed at both extremities, the right-hand finger will be stimulated twice as often during the same time as the left, giving a difference which may be compared with the musical octave. Intervening points—not shown in the cut—are placed on the board, corresponding to thirds, fourths, &c., down to a quarter of a

tone. In practising with this instrument the operator must be blindfold, and another person should be present in order to reverse the position of the touch-board. This is absolutely necessary, for the imagination has much power in this kind of experimentation. In my own case it is perfectly easy, without a previous experimental course, to distinguish the intervals down to a semitone. I do not think I am more sensitive to roughness—differences—than others who use their fingers. No doubt those engaged in many trades, where there is constant testing of the roughness of materials, could distinguish smaller intervals. In the case of the blind, too, this sense is of great importance. I have not yet tried a blind person with the touch-board, but I should expect, that in this case very small intervals indeed could be detected.

In music, we find that small intervals are not distinguished so readily in some parts of the scale as in others. Thus towards the extreme upper limit, one may be unable to distinguish two notes a semitone apart. It is the same with touch. If the hands are drawn very rapidly across the board, the smaller interval cannot be made out. Towards the extreme upper limit I am able, however, to distinguish an interval of a third.

The sense of roughness, to which so little attention has been given, is yet of great importance. It has qualitative variations, almost as great as the musical scale, and the sensations are definite, and may be distinguished fairly sharply one from another. It is by this sense of roughness that we learn much of the nature of the external universe. By it we distinguish the roughened border of a file, the granularity of a surface, and the texture of a fabric. Its study has not engrossed the minds of physiologists and psychologists, because of the prevalent idea that it is merely an intermittent sensation, resulting from intermittent stimulation. This we have shown not to be the case.

Let us turn to the larger question, namely, that quality of sensation is objectively determined by frequency of stimulation. In the senses of sight and hearing this is undoubtedly the case. I mentioned that strong evidence has lately been brought forward in favour of smell, and some experiments of my own on taste tend towards the same result. In touch, too, we have

a curious example of a sense where subjective quality evidently depends upon objective frequency of impact. A curious negative proof is furnished in the case of temperature. Here we cannot be said to have any subjective quality at all—merely a sense of quantity. On studying objective temperature we find that the molecules of bodies, when heated to different temperatures, do not vibrate at different rates, but only at different amplitudes. Here we have no variations in objective frequency, and no variations in subjective quality.

SENSATION AND MOVEMENT.

BY CH. FÉRÉ, M.D.,

Assistant-Physician at the Salpêtrière, Paris.

IN a series of communications made this year to the Société de Biologie and to the Société de Psychologie Physiologique of Paris, I endeavoured to show that peripheral excitations and psychical operations are accompanied with motor phenomena which, under favourable experimental conditions, may be demonstrated even by rough methods. These facts, which give a material demonstration of some views expressed by modern psychologists, may be found worthy of being brought together here in a condensed form.

The naturalist Péron had (1800–1804) found that the aborigines of New Holland, and the Malays of the island Timor, displayed a much smaller power of muscular effort than the French sailors with whom they were compared. Dr. Manouvrier¹ made the same remark concerning the majority of the savage tribes exhibited in the Jardin d'Acclimatation. I have also observed in a certain number of negroes that the muscular expression on the dynamometer is inferior to that displayed by most Europeans.

Broca had undertaken, during the latter part of his life, researches which, though not found mentioned in his published works, have formed the starting-point of our own researches. They dealt with the strength, as tested on the dynamometer, of persons belonging to various classes of society. The result was that the pressure produced by the effort of closing the fingers is less among workmen whose profession is solely manual, than among those who spend less muscular force, but whose intelligence is brought into use; and that it is greater still among people belonging to the liberal professions, under

¹ "La Fonction psychomotrice;" 'Revue Philosophique,' June, 1884.

similar conditions of age. The influence of body-height is not considerable. A certain number of experiments show, that among women, the greatest effect of a momentary effort is related to the greatest intellectual activity.

Dr. Manouvrier has made dynamometric researches on subjects who do not professionally exercise their muscles, and thinks that the energy of the contraction may be related to the size of the brain; but there is no proof of this assertion. We may assume, however, that *the energy of the momentary effort corresponds with the habitual exercise of the intellectual functions.*

It may be objected that it is impossible to determine the part played by differences of alimentation and hygiene in the production of differences in the energy of voluntary movement; and moreover, that it is imprudent, in such matters, to frame definite conclusions from insufficient statistics. It was therefore necessary to bring forward new facts in support of this influence of intellectual work.

It is generally known that under the influence of certain physiological states, such as anger, or of pathological conditions, such as maniacal excitement, muscular efforts acquire an extraordinary vigour; but this exaggeration has never been regularly measured. Dr. Manouvrier had already remarked that the fact alone of experimenting in public increases the strength of the movements; this is true, and we may add, that the experiment made before a person of the opposite sex exaggerates it still further. This excito-motor influence is, I believe, proportionate to the excitability of the sexual feelings, and deserves to be studied more fully from the point of view of selection.

These facts are interesting, but insufficient to explain the part played by psychical action, which must be put into evidence by another method. It is to be noticed at the outset that one obtains fairly constant dynamometric results on the same individual and with the same instrument, and that exercise has but little influence. I have made upon myself many thousand experiments, divided into series at several months' interval, and have noticed but a very slight and gradual increase. It was important to settle this point before

investigating the daily alterations due to the influences we have to study.

Under the influence of intellectual work, the dynamometric readings show an increase of one-sixth, one-fifth, or even one-fourth, according to the kind of work, and according to the more or less steady attention given to it. In many explorations I have observed a tendency to an equalisation of both hands—*i.e.* that the left hand (which is weaker than the right by 10 kilos nearly) often gains more rapidly than the right under the influence of the psychical excitation due to the intellectual work. This increase is momentary, and generally disappears a few minutes after the stimulus has been removed. These experiments, which show that *at the moment of intellectual activity there is a momentary increase of voluntary power of movement*, strengthen our first conclusion, and explain the fact already observed, that dynamometric measurements taken immediately after rest, generally point to a less degree of power for muscular effort than those made later, after the psychical functions have been exercised.

In order to appreciate the dynamogenic value of psychical functions, we must study only such operations as are accompanied with the least possible movement, such as hearing a speech, silent reading, &c.; for the act of speaking and writing introduce a new motor element into the experiment.

It is not only under the influence of an intellectual effort that the dynamometric power is augmented; the exercise of a limb different from that under observation may produce an analogous effect, though less markedly. If, for instance, one makes with the foot the movements necessary to drive a wheel with a pedal, it is observed that in a short time the dynamometric power of the corresponding hand, then that of the other, is augmented by one-sixth or more. The exercise of speech may produce the same effects, and its influence on the psychical excitation is well marked in certain nervous individuals, who become, as it were, intoxicated by speaking; thus we see that the most excito-motor psychical acts are those which are accompanied with phonetic or written signs, or expressive movements.

On the other hand, if one makes during one or two minutes a series of movements of flexion with the hand to be tested, an

augmentation of the force of the dynamometric pression will be observed. This fact is in accordance with the remark made by most experimenters, that the second squeeze on the dynamometer is usually stronger than the first. The instinctive habit of stretching the limbs has for its effect an awakening of the muscular sense. These experiments show, that when a limb is put into action the movement determines in the cerebral centre of this limb dynamogenic action, which spreads to the neighbouring centres. We may perhaps infer therefrom, that the paralysis of the same centre would be capable of exercising an inhibitory action; and thus might be explained the fact, that even when no bilateral descending degeneration is present, every destructive lesion in the motor region produces a weakening in the four limbs. Brown-Séquard has insisted upon this view more than once.

Finally, we may say, that passive movements produce the same motor excitation as active; the excitation appears to be in the former case even more intense. This result acquires considerable importance when taken along with the following facts. The history of spasmodic epidemics shows, that among individuals sensitive to all dynamogenic and inhibitory agencies, the sight of a rhythmical movement may excite the production of this movement. This phenomenon of psychomotor induction may be sporadic, as Prof. Ch. Richet has shown. If the subject be made to look attentively at the movements of flexion which we make with our hand, after a few minutes he says that he has the sensation of the same movement in his hand, though it is quite motionless; and after a little while, his hand begins to execute, irresistibly, rhythmical movements of flexion. Now, if instead of going so far, we stop when the subject begins to have the sensation only of the movement, when the latter is in the nascent stage, we place a dynamometer in his hand, it is found that the force of pressure is augmented by one-third or one-half. These facts seem to us to show, that *the energy of a movement corresponds with the intensity of its mental representation*. This deduction corresponds with the views of psychologists, who state that the idea of a movement is already the beginning of the movement. Hence it follows, that whenever the idea is

sufficiently vivid, the action follows necessarily. This is an important point in explaining irresistible impulses, when the act is the inevitable consequence of the persistence of the idea. On the other hand, we may say that an idea really exists only when it is followed by an act which is the only proof of its intensity. We must distinguish between weak or static ideas, and strong or dynamic ideas. The excito-motor influence of passive movements is precisely due to the fact, that they provoke a recal of their motor images.

This influence of passive movements is taken advantage of in the treatment of certain cases of loss of power. When single muscles have been excited, either by massage or by electricity, the function of each muscle may be re-established by an action on the psycho-motor centre; but the function of the limb may nevertheless remain in abeyance, owing to the absence of co-ordinating impulses; the latter are sometimes quickly restored by the exercise of artificial movements, which complete the re-education of the centres.

The central effect of peripheral excitations may be placed into evidence by other experimental facts observed in persons subject to hypnotism, and upon whom dynamogenic agencies have a more definite influence. If on one of these subjects, when awake, a muscle be excited by massage, it becomes rapidly tetanised; the repetition of a passive movement which brings the same muscle into play, produces the same effect; voluntary action has the same result: the identity of this result shows the fundamental identity of the process. When a muscle is excited by some means or other, its motor centre is acted upon by the revival of a motor image. If the excitation is intense, tetanisation spreads to the associated muscles, and even to all the muscles of the limb, and when a fresh excitation is made on an antagonistic muscle, an epileptiform convulsion is often produced, which may bring about loss of consciousness, and become generalised through the whole body. This succession of phenomena can be explained only by an excitation of the higher centres, which is produced the more readily the greater the psycho-motor excitability of the subject.

I have, up to this point, been endeavouring to bring into

light the influence of psychical stimulation, whatever be its origin, on the energy of voluntary movements, and particularly on the momentary effort. The sustained effort, *i.e.* the resistance to fatigue proceeding from a kind of automatism, is different, and deserves a separate study. I may mention that if psychical activity has an influence on the energy of voluntary movements, the latter may also influence the former. Some people instinctively begin to walk when they wish to concentrate their thoughts, and the effect of movement is sometimes sufficiently marked to be appreciated by them; an increase of the power of recollection has more particularly been observed.

We may conclude, that each time one of the cerebral centres enters into action, it determines an excitation of the whole by a process which has not yet been determined. This statement is important from the point of view of hygiene and education, as showing the use of bringing into play as many functions as possible in developing all and each particular function.

Most of the preceding conclusions have been derived from experiments made upon myself, but certain of these results required further differentiation. Hence, I resorted to subjects presenting a morbid excitability, and so to speak, an enlargement of dynamic manifestations. Hysterical individuals, and particularly those liable to hypnotism, are particularly good subjects, and are for the purposes of the experimental psychologist what frogs are to the physiologist. Here is a series of experiments made on such a subject.

The patient was one of Professor Charcot's hystero-epileptics. She has double anæsthesia, chiefly on the left; the dynamometer shows an average of 23 kilos on the right, and 15 on the left. These are low figures, considering her robust appearance; but we know that hysterics usually have a weakness of the muscles, chiefly on the anæsthetic side. The experiments were made at various intervals, and always after a preliminary dynamometric exploration, which showed that her average state was not modified. The constancy of the results eliminates every fallacy arising from the action of the patient's will. The following numbers are clear enough.

		RIGHT.	LEFT.
(a)	Normal dynamometric strength	.. 23	.. 15
(b)	After 20 passive movements of flexion of the right fingers	.. 41	.. 14
(c)	After 20 active movements	.. 45	.. 20
(d)	After having counted up to 45..	.. 44	.. 24
(e)	After mental effort of adding 366 and 374 41	.. 36

These numbers show that when intellect comes into play, dynamogenic action tends to be diffused to the opposite side ; and that in the intellectual effort there is a certain tendency to the equilibration of the strength of both hands.

	RIGHT.	LEFT.
(f)	After 20 movements of the right leg (voluntary flexion of the two segments)	.. 46 .. 28

This experiment shows, that the exercise of a leg exerts its chief dynamogenic action on the arm of the same side ; this action is even sometimes limited to that side. The synergy of the two limbs on the same side is still further shown by the fact, that when an effort is made with the right hand, it is in the right leg, and particularly in the triceps, that a sensation of contraction is experienced, and this contraction sometimes becomes real.

We may perhaps legitimately conclude from this fact, that the exercise of one limb dynamogenises the other by calling forth, in its psychical centre, motor representations which gradually become diffused to the opposite side. We may give numbers which will confirm what we have been saying, that the development of motor strength is under the influence of the mental representation of the movement :

(g) The right hand of the patient is extended on the knee, the experimenter places his own hand near, fixes upon it the attention of the subject and repeats 20 times the movement of flexion of the fingers ; at this moment, the dynamometric strength of the right hand is 46 instead of 23 ; it is doubled, whilst that of the left hand is a little diminished—12 instead of 16.

This diminution of the left side shows, that when the attention is intent upon one limb, a kind of compensation is made at the expense of the others. The same effect is noted when it is suggested to a somnambulist that he is endowed with great strength in one limb : that of the opposite diminishes. Conversely, when a psychical paralysis of one limb is produced,

the strength of the other is increased; there is not so much a loss as an alteration of distribution. If this characteristic was general among hemiplegias by suggestion, it would supply important data for diagnosis; for we know that in hemiplegia through gross cerebral lesion, there is, on the contrary, diminution of the strength on the opposite side.

I imagined, from certain observations, that sexual excitement did produce a momentary increase of dynamometric strength. The investigation of this point led me to the discovery of some interesting points. Dr. Chambard has shown, in some somnambulists, the existence of "erogenic" zones, irritation of which produces feelings of congestion of the generative organs, and erotic ideas. These zones, which are found chiefly on the neck and on the neighbouring portions of the thorax and head, do not lose their properties during the waking states; they exist in persons who have never fallen, or been placed, in the somnambulistic condition, and are even, perhaps, present, up to a certain point, in everybody. I tried to find, by exercising on them a slight pressure, whether these excito-genital zones were at the same time excito-motor, and found the dynamometric readings to rise to 38 or 40 on the right side, and to 32 or 36 on the left, according to the zone, cervical or sternal, excited. I likewise explored the hysterogenic zones on the same subject, and obtained similar results. I further discovered purely dynamogenic zones where the most energetic pressure produces nothing more than an increase in the dynamometric pression. Two such zones, placed laterally behind the bregma, or superior fontanelle, on each side of the median line, exercise a crossed action upon the hands, the dynamometer on the right side ascending to 42, on the left to 35 kilos.

These facts, which I merely mention here, seem to indicate a transition between the dynamogenic and the convulsing actions (hysterogenic and epileptogenic zones); the latter effect being only an exaggeration of the former. We see how dynamogenic excitations, such as intellectual exercise, may determine, when exaggerated, distinctly neuropathic states, susceptible of hereditary transmission. This is exactly what occurs in the case of the guinea-pigs, which, when made

epileptic through peripheral excitations, transmit this neurosis to their descendants. I shall, on a future occasion, return to the pathogenic influence of "mental overpressure" (surmenage intellectuel).

The very definite results, which are obtainable in hysteria, may sometimes be produced with the same intensity on healthy subjects, in whom fatigue has determined a state of irritable weakness, and thereby a hyper-excitability analogous to that of hysteria. Under the influence of fatigue, due to protracted intellectual labour, dynamometric force diminishes and tends to become equal on both sides. Let us suppose in the normal state 55 on the right and 45 on the left, we should find, under the influence of fatigue, 40 on both sides. If now we carry out passive movements of flexion of the hand, the dynamometer may show 60 on the right and 50 on the left; the strength of contraction has been brought up to the same degree as if the excitation has been made on the normal state; but its relative effect has been much more considerable. Hysterical persons are in a permanent state of physical fatigue, but various excitations may temporarily awaken their energy, as it may do also in neurasthenia.

I have often found that, under the influence of fatigue localised in the right hand, for instance, the left showed, on the contrary, an increase of one-fifth. This fact may serve as a link between paralysis by exhaustion and certain paralyses by suggestion, in which we have found that the dynamometric strength is increased on the opposite side. In confirmation of this view, we may state, that as passive movements do counteract the diminution of contraction due to general fatigue, so certain paralyses by suggestion may be cured by the same movements. This remark may perhaps be of use in the treatment of hysterical paralysis, where psychical alterations play such an important part.

We have already said that the act of *speaking* could produce an increase of dynamometric strength on the right side. Excitation in the opposite direction may be illustrated in a case of motor aphasia induced by suggestion. If in such a case we practised passive movements of all the segments of

the right arm, we should find that after a moment the power of speech returns, but disappears again as soon as the movements of the arm cease; the same result is obtained if the subject performs active movements with the right arm. This observation, which may perhaps also find an application in the case of certain hysterical aphasias, shows how the exciting influence of such movements is exerted on the movements of the organs of speech. It may also explain the persistency of the mimical movements of the limbs, which, though often without significance of their own, may nevertheless act as an excitant to the act of speech. Finally, it shows that the fact of the functional predominance of the right arm (of which the motor centre is on the left side), and the localisation of the function of language on the left side is not a simple coincidence, but that there is a cause for this relation. It is because the child has chiefly used his right arm, whether it be through instinct or education, that he speaks with his left brain.

I have observed that, generally speaking, psychical excitation is accompanied with an increase of motor energy, appreciable on the dynamometer when the experimental conditions are favourable. I have noticed, also, that certain peripheral excitations of the muscular sense have an analogous effect, which remains localised or becomes diffused according as the excitation itself has been more or less limited and pronounced. The facts relating to the special zones have shown the results of certain excitations of the general sensibility; and, finally, in the sexual sphere, we have observed effects of the same description. I have noted, by the way, that the hallucinations induced in hypnotised individuals have the same property of increasing the dynamometric force, whatever be the sense involved. When the hallucination is strictly unilateral, dynamogeny exists on the same side only—a fact, by the way, which gives a new proof of the reality of these induced hallucinations.

The existence of this dynamogenic property of hallucinations has led me to seek whether some analogous fact was not connected with the simple excitation of the several organs of sense. I discovered that a strong stimulus of sight,

hearing, smell, taste, or touch, determine in the healthy subject a notable increase of the deflection of the dynamometric needle; the reaction varied with the intensity of the excitation. I draw the conclusion, that *the impressions made upon the various senses have a common measure*, supplied by the dynamometer; all sensations are accompanied with an augmentation of potential energy, which appears to constitute the essence of a sensation. These facts, moreover, are in perfect harmony with the mode of embryonic development of the organs of sense, which have a common origin, but show their differentiation to be less complete than would at first sight appear to be the case.

Neuropathic, especially hysterical, persons, who normally present a certain degree of anæsthesia involving the muscular sense and a corresponding amount of weakness, display these phenomena on a magnified scale. On such a subject one may see the dynamometric power doubled under the influence of a vivid sensorial excitation; and memory, which is but a recall of the sensation, has, when intense, a similar effect. These facts show that *psycho-physiological functions are reducible to mechanical work*.

My experiments show, that under certain favourable circumstances the dynamometer may become a measure of sensation: I shall return to this point.

I have previously seen that, if a certain degree of cerebral excitation develops muscular energy, intellectual fatigue diminishes it; excitations of sensory organs have a similar result. If I tell a hypnotisable person to look at a moderately luminous object, a motor excitation is at once produced, which after a few instants decreases when the subject begins to complain of fatigue; if the excitation be continued, sleep supervenes. A continuous sound or mechanical vibration, &c. produces exactly the same effects, and in the same order of succession. When, instead of moderate and prolonged, the excitation is sharp and short, sleep may supervene at once. These facts are in accordance with those which Brown-Séquard has grouped under the appropriate names of *dynamogeny* and *inhibition*. They show that peripheral stimuli may produce, according to their intensity and duration, exciting or

depressing effects which may culminate into convulsions and paralysis.

When I consider separately the effects of stimulating each of the various sensory organs, I come to conclusions which are not without interest and points of concordance with one another. With reference to hearing, for instance, we find that sounds have a dynamogenic influence which varies with their loudness and their pitch; in other words, that *the intensity of auditory impressions, measured by their dynamic equivalent, is in relation to the amplitude and number of vibrations*. When I speak of these impressions I mean the uniform, monotonous sensations due to a single note. The compound auditory impressions constituting harmony, and to which memory and association of ideas may give a peculiar meaning, have a variable action according to circumstances. Hence we find different results according to whether we deal with lively or sad music; the latter is depressant, the former excito-motor. These effects, which in healthy subjects are readily made manifest on the dynamometer, support the conclusion which we shall deduce from further facts, that sensations are agreeable or painful according as they augment or diminish the potential energy. At any rate, it shows that it may be of importance to exercise care in selecting the pieces which children are made to play.

There is some reason in the idea long entertained, that music has an influence in the treatment of mental disease, but we see that its use must be based upon certain principles; it acts not as rhythm only, as some writers have asserted, but as sound in itself. We are at present engaged in a detailed study of the question of auditory impressions.

With reference to vision, the results are no less interesting. Observations on the hallucinations of hypnotised individuals have already shown, that colours are susceptible of being classified, according to their dynamogenic effects, into a certain order, which appears to be—red, orange, green, yellow, blue. Experiments made on real sensations have given similar results. The effects of the more active colours are very definite in many people, but more especially in nervous subjects. Thus, on the subject already mentioned, whose normal dynamometric power is equal to 23 kilos, we find that

when coloured glass or gelatine plates are used, red rays bring the pressure up to 42, orange to 35, yellow to 30, green to 28, blue to 24.

It will be observed that in these experiments on real sensations, yellow does not occupy the same position as it does in hallucinations. This deviation is highly instructive, for in some recent experiments made with M. Londe, in which we operated with the spectral colours by decomposing white light through a prism of sulphide of carbon, we found that the dynamic effects of coloured rays diminish from red, to orange, green, blue, violet; those of yellow being similar to those of blue, and much less marked than in our previous researches. The error in the first instance came from the fact that the so-called yellow glasses were really of an orange tint. It is interesting to note that, in the case of the hallucinations of hypnotised subjects, the colour suggested to the latter was of a perfectly pure tint.

It thus appears that, with this exception, the dynamogenic power of colours follows the same order as their spectral position; hence *the intensity of visual sensations varies with the vibrations*. The *vibration* therefore appears to be the hint of excitation as well for the sense of hearing as for that of sight. Does the same law hold in the case of the other senses?

With reference to the predominating influence of the red, it is interesting to remark, that it is the colour which seems to have most effect upon animals, and to be the first distinguished by children; the first also to have been recognised by man. Violet, which exerts a depressant action, has been used, as well as blue, with a certain success in calming maniacal excitement. Finally, we may add that special experimental dispositions have enabled us to ascertain, that for each colour the dynamogenic action varies with the intensity of light.

We have studied taste by the same process, and found that its fundamental sensations may be classified according to a dynamic scale, analogous to that of sounds and colours. Thus, sugar has a very low dynamogenic action; salt, a more marked action; bitter substances are the most active. Thus, on the hysterical subject of our previous experiments, the dynamometer

after sugar gives 29 kilos; after salt, 35; after quinine, 39. On normal persons, though less marked, the action of salt and bitter may be quite manifest. Acids are still more powerful, but their action is complex. General sensation and smell are affected at the same time, and their action is added to that of the sensation of taste. Acetic acid, for instance, is much more active than bitters.

The effects of sugar, &c., in solutions of definite strength show that they augment with the concentration, though not susceptible of being reduced to a mathematical formula. The dynamic condition of the subject under observation varies under the influence of the ingesta, &c. But if we cannot formulate a psycho-mechanical law, the general data are sufficient for approximations.

The dynamic influence of the sensations of taste, especially when associated with those of smell, may serve to clear up a point of physiology still controverted. There is a food, beef-tea, of which the nutritive value is contested, chemical analysis showing that it contains but a slight amount of, if any, alimentary material. In its defence, the view is taken that it must have some value, because the patient under its influence often regains some strength, and becomes capable of a certain momentary effort. It is certain that the action of this aliment on taste and smell may determine an excitation of strength, especially in an exhausted subject, for we have shown that under conditions of exhaustion, dynamogenic agents have a more marked influence. Beef-tea, as a sensorial stimulant, has a dynamic effect. The smoke of tobacco produces the same effect on certain individuals. This influence no doubt extends to the digestive organs, and may account for the eupeptic action. Certain bitters, considered as aperients, have a contrary action, on account, perhaps, of the over-excitation which they exert, either by themselves or by the substances which are associated with them.

Our studies of the olfactory sensations are less advanced. Still, we have been able to establish a kind of dynamogenic scale of smells, in which musk appears to occupy the highest position.

It may be said in a general way, therefore, that *all sensations*

determine the development of potential energy which passes to the kinetic state, and manifests itself by a motor action, capable of demonstration even by such rough procedures as dynamometric measurement.

The excitations of internal organs may determine similar dynamogenic actions. Thus, squeezing even lightly the os uteri, which in the normal condition is insensible, may determine an augmentation of dynamometric pressure. This observation shows that the stimulus need not be perceived in order to determine a mechanical action. This is an interesting point with reference to convulsions arising from non-painful visceral lesions; and as an illustration of this point, we may allude to a series of observations on the effects of the excitation produced by coloured rays on an achromatopsic eye when the dynamic effects were found to be parallel to those observed on the healthy eye.

Hence we may conclude, that *every peripheral excitation determines an augmentation of potential energy*. The consequence of any excitation whatever, whether of a physical or chemical nature, is a transformation of force, a modification of a form of movement. We have seen more particularly that for sensations of sight and hearing, there is a relation between the amount of stimulus and the amount of reaction, the strength of movement produced giving the measure, up to a certain point, of the initial vibration. The human organism, though so complex, thus reacts as would any other body to external action. Nothing but transformations have taken place, of which, it is true, many escape our means of analysis. This transformation of movement, as observed in the simplest organisms which react by an appreciable change of form, constitutes the essential function of nerve elements. Whether we deal with a simple reflex or a complex psychical operation, everything becomes reducible to a dynamic transformation which may be made evident by the exploration of the motor residua of cerebral action.

The human body behaves, in a word, like any other mass of matter which transmits a communicated movement, transforming it, however, according to its own molecular constitution. This constitution of living organisms however, or in

other words, the form of their peculiar vibrations, varies continually with the processes of assimilation and disassimilation. Each individual and each part of the individual reacts according to its specific energy, and the individual variations explain how every subject may transform differently a communicated movement, and how he may react differently to the same stimulus, according to circumstances. Some react chiefly to olfactory excitations, others to auditory, &c. We need not therefore have recourse to the theory of the perigenesis of plastidules and of plastidular vibrations, to account for these differences; direct observation is enough for their explanation.

The form of the vibratory movements of the body is modified every time it is placed in contact with another vibrating body. These alterations in the form of its vibrations account for the changes in the dynamic state observed under the influence of light, sound, &c. The vibrations of the diapason determine in certain subjects considerable functional modifications; magnets act in the same way, and metals according to their atomic constitution.

It is only by these modifications of the vibratory form of the body elements, and especially of the nervous system, that we can explain the phenomena of dynamogeny, of inhibition, of transfer, of psychical polarisation, which occur without any appreciable material modification.

The influence on the dynamic state of the vibratory form of the forces which come into contact with the external integument is experimentally shown by some of the facts previously described. We think ourselves entitled to conclude, that *psycho-physiological functions are reducible to mechanical work*. Mechanical work is easily recognised in the simple reflexes of inferior organism, but its study is more difficult when we deal with the complicated reflexes constituting the psychical operations; the process, however, is always the same. If we have insisted at such length upon these considerations, it was to establish once more that psychology of cerebral physiology must be studied by the general methods of biology. Experience is to form the basis of all natural sciences, and an effect is not to be denied because we do not understand its mechanism. We have experimentally shown that every stimulus, even when

not perceived, determines a dynamic effect. This result is of physiological, as well as pathological and therapeutical importance. Thus with reference to external irritations of revulsive actions, of which the influence is denied or exaggerated according to the rationalistic or empirical bias, our experiments may furnish a possible explanation. They show that the dynamic effects of peripheral stimuli are exerted, not only on voluntary muscles, but also on those of organic life; the augmentation they produce in the circulatory activity, for instance, is very clear, and long ago Haller observed that the sound of the drum increased the flow of blood from an open vein. Such an influence on the circulatory and nutritive activity makes plain how a blister, for instance, may determine nutritive phenomena, absorption of liquids, &c., with predominance on the same side of the body, but is capable also of acting at a distance. These circulatory effects of peripheral stimuli may also assist us in the interpretation of certain psychical phenomena.

I shall have occasion to return to the subject of motor manifestations in muscles of organic life under the influence of external excitations; but I may mention here that Majendie, Cl. Bernard, François-Franck, Couty and Charpentier, and others, have spoken, though with some divergences of opinion, of the cardio-vascular effects of sensory excitations. On the other hand, Cl. Bernard, Schiff, Vulpian, &c., have shown that the pupil contracts under the influence of exciting sensations; and certain emotions, such as anger (Jorissenne), have the same effect. The pupil dilates under the influence of pain, and depressant emotions such as terror; the same phenomenon is noticed in fatigue. Finally, in their experiments on the bladder, Mosso and Pellacani have found that excitation of any sensory nerve, as well as psychical activity, produce reflex movements of that organ. I may add that voluntary contractions of the anus, as recorded by a graphical process which need not be described here, are more energetic and durable under the influence of certain sensorial or psychical excitations. Hence we may conclude, that *every impression augments the energy of the whole organism.*

My more recent researches with the dynamograph have

fully confirmed all these results, and show in addition that the muscular contractions effected under the influence of excitations of the several senses differ, not only in intensity, but also in respect of their dynamographic curve.

As Spencer remarks, the phenomena of pleasure and pain are perhaps the most obscure and complex in psychology. It may, therefore, prove interesting to bring forward certain facts calculated to throw light upon their genesis.

I have already mentioned, with reference to auditory excitation, that painful sensation is accompanied with a depression, whilst agreeable sensations coincide with an increase of energy. I may add that, under the influence of pleasant or unpleasant emotions arising from any other cause, the same dynamometric variations are observed. When a subject is submitted to the process elsewhere described as *psychical polarisation*,¹ and which consists in a kind of inversion of the psychological condition under the influence of an external stimulus, the dynamic state is altered along with the emotive state. Conversely, it is manifest that colours or smells which will produce the most intense dynamogenic effects are as a rule pleasant. I have reached the conclusion, that agreeable or disagreeable sensations depend upon an increase or diminution of potential energy. Certain facts, however, relating to painful feelings seem to make an exception. The investigation of certain olfactory sensations may re-establish a concordance amongst these apparently conflicting data.

Dr. G., who is very sensitive to the action of smells, kindly consented to be experimented upon. After having found that the dynamometric pressure of the right hand varies from 50 to 55, we rapidly placed under his nose a bottle containing perfectly pure musk. Dr. G. declares the smell to be extremely disagreeable, his dynamometric force now amounts to 45, and therefore is diminished. The same experiment was repeated later on, but with the bottle held at a distance, so as to attenuate the impression. He now declares the smell to be pleasant, and his physiognomy clearly expresses satisfaction. His pressure now is 65; in other words, is augmented by 10 or 15 kilos.

¹ Binet and Féré,—‘Revue Philosophique,’ April, 1885.

In a hysterical patient who is anæsthetic, and shows a considerable loss of smell, the close contact of the musk determines a very agreeable sensation, and at the same time considerable dynamogenic effects (46 instead of 43). In another experiment on the same subject, the bottle was left in contact with the nostrils for three minutes. The sensation, after being pleasant at first, began to cause inconvenience, and the dynamometer now showing 19; in other words, a marked diminution. On keeping up the action of the musk, sensation becomes weaker, disappears, dynamometric action becomes lower, and finally the subject falls into a lethargic sleep. The olfactory sensations act exactly as those of hearing and sight; they all are exciting at the beginning, then produce exhaustion, and sleep in predisposed individuals.

This succession of phenomena shows that artificial sleep does not originally differ from normal sleep; fatigue is its primordial cause. This fatigue may be due to a sudden discharge of reflex movements determined by sudden stimulus, or is brought about slowly in consequence of a prolonged and monotonous impression. A similar sequence under the influence of one stimulus (strong sensation, disappearance of sensation, fatigue) is physiological, and may be observed in the case of many persons. It shows that an excitation, even when no longer perceived, still determines dynamic effects and fatigue. Unpleasant stimulus may cease to be felt without ceasing to exert mechanical effects, the absence of which is recognised when the stimulation ceases.

These various experiments coincide to show that pleasant sensations are accompanied with an augmentation, and unpleasant sensation with a diminution of energy. The sense of pressure resolves itself, therefore, in a feeling of power; that of pain in a feeling of powerlessness. Hence, we have here a material demonstration of the theoretical views more or less clearly expressed by Kant, Bain, and Darwin, upon pleasure and pain.

Now every stimulus which augments potential energy ends in a discharge; now gradual, when the stimulus is moderate, now sudden, when it is strong and determines reflex movements. The impression of ammonia on smell determines such movements; it produces a sudden discharge with rapid

depression of potential energy ; hence a disagreeable sensation. The immediate and momentary increase of the dynamometric pressure is only the direct effect of the reflex discharge. Each discharge is accompanied with a diminution of potential, so that, beyond a certain limit, sensation ceases to grow proportionally to the excitation.

We have already seen that auditory impressions are followed by analogous phenomena of increase and diminution. Other sensory impressions do the same ; those of sight show not only that the fatigue of an organ depends on a diminution of the potential energy of the subject, but also that this diminution coincides with a modification of the molecular vibrations. When one fixes the eyes for a long time upon a red square on a white background, green appears on its edges ; this is the phenomenon of simultaneous contrast ; on weak subjects, and in the state of fatigue, the phenomenon occurs more quickly ; and it may be produced at once in certain persons by the application of a magnet, which acts by modifying by its own vibrations the molecular vibrations of the body.

Our chief object, however, was to show that pleasure and pain are correlated with the potential energy of the subject. The senses of sight, smell, hearing, taste, are not the only ones which demonstrate this fact ; excitations of the genital sense are no less demonstrative in their effects. It is easy to account on these principles for the exaltation of energy, which grows to a paroxysm, and is followed after a while by a persistent depression. We need not insist upon the psychical states which correspond to this dynamic exaltation and depression.

These are remarks on the bodily phenomena which accompany pleasure or pain ; the latter, being but a mode of fatigue, may serve as a basis to a physiological theory of æsthetics. And as pleasure and pain are at the root of what are known as sentiments, affections, elective affinities, these psychical states will also find their physiological explanation in the same considerations.

In a future contribution we shall bring new facts, furnishing a physical demonstration of the view that the living being does nothing but to restitute, under another form, communicated movements.

NERVE TROUBLES AS FORESHADOWED IN THE CHILD.

BY CH. FÉRÉ, M.D.,

Assistant Physician at the Salpêtrière, Paris.

WE have elsewhere¹ adduced arguments tending to show that heredity is the primordial cause of nervous affections, whether they be characterised by somatic or by psychical disturbances. We have adduced a certain number of observations enabling us to generalise the views of Morel, of Moreau (de Tours), of Prosper Lucas, of Trousseau, of Griesinger, of Charcot, concerning the ætiology of these diseases.

But it must be admitted that in many cases it is impossible to follow up the hereditary evolution of the neuropathy, which may further be due to some disturbance in the conception, in gestation, or in the first childhood. No one doubts to-day that children conceived during a drunken debauch are peculiarly predisposed to idiocy, imbecility, epilepsy, &c. The passions, the emotions, may play the same part : a fact of which we have related an interesting example. Certain disturbances of body or mind during the course of gestation may have identical results, as shown by the statistics of the children born during the siege of Landrecies ; and Legrand du Saulle has made recently interesting observations with reference to the siege of Paris. During early childhood defective hygienic conditions may sometimes influence the development of the nervous system, and take a share in the causation of certain more or less indelible pathological states. Thus, a prolonged lying on the right side may determine certain deviations of the form of the skull by lateral propulsion (Gueniot), which are the more readily produced if the young subject is at the same

¹ "La Famille Névropathique—Eclampsie et Epilepsie;" 'Archives de Neurologie,' 1884.

time affected with athrepsia (Parrot); and functional troubles of variable severity may result from this cause.

When no trace of degenerescence is discovered in the heredity of a subject, it does not follow that the nervous affection from which he suffers is accidental, arising from some purely fortuitous cause, such as a traumatism, or an infectious disease, &c. The patient may not have any hereditary taint, but be the victim of a disturbance of evolution which may not break out until the development of the nervous system has reached its completion.

The predisposition may thus result from an hereditary degenerescence, or from an evolutionary defect giving rise to weak points which account for the systematisation of certain lesions, the localisation of the latter being determined by the defective evolution of the affected parts.

Hence, before we can admit the accidental origin of a nervous affection, it is necessary to eliminate not only the presence of any direct or indirect hereditary condition, whether similar or transformatory, but also that of any disturbance of evolution capable of bringing about a defective development of the nervous system.

To sum up: Before we speak of the accidental origin of a neuropathic affection we must make sure that the ancestors, and the subject himself, have never presented any nervous trouble. In order to determine the existence of the alterations which form the substratum of a morbid nervous predisposition, two orders of facts must be taken into consideration: some relate to organic changes, the others to functional troubles to which no certain anatomical basis can be ascribed.

Morel first showed that the insane were, more frequently than others, subject to bodily malformations. This remark has been confirmed by many observers, and we may now generalise the remark, and say that this tendency to degenerations is not peculiar to them, but is common to all subjects with a neuropathic disposition.

The anomalies of development may affect the whole organism, and thus men whose muscles, hairs, generative organs are little developed and present general analogies to a woman,—presenting in other words the feminine type

("Féminisme")—are very liable to various nervous accidents. A certain number of males suffering from hystero-epilepsy, for instance, present this aspect.

Conversely, women who by their external forms, their muscular development, the scanty volume of their mammary glands, their exaggerated hair-growth, especially in the sub-maxillary region, have virile characteristics ("Masculisme"), are predisposed to neurotic affections. Such women frequently suffer from the newly described forms of alienation which are peculiar to those in whom we recognise hereditary tendencies to degeneration.

A certain number of subjects of both sexes present a retardation of development (called "*Infantilisme*" by some authors), in consequence of which all the organs and tissues are more readily attacked by degenerations and infections. Lorain used to lay much stress on the frequency of tuberculosis among such subjects. It is to be noted that such subjects usually reach early the stage of decrepitude, and often show the characteristic features of *precocious senility*. The laws of evolution govern tissues and organs as much as individuals and species. We have shown¹ that the senile atrophy of bony tissue, for instance, shows itself first in the regions of most recent growth, and of most laborious development, and that these regions have the greatest tendency to become the seat of various pathological lesions. It is a well-known fact, that organs which have been late or disturbed in their evolution are more exposed to inflammations and organic degenerations; the pathology of the testicle, in particular, furnishes numerous examples of this kind (Le Double, Aubert). The nervous system makes no exception to this rule. A good many of the individuals affected with infantile spastic paraplegia are born before the term.

The anomalies of formation which stand in closest relationship to affections of the nervous system are those relating to the cerebro-spinal centres and their envelopes. We need not insist upon such malformations of the nerve-centres, which

¹ "Atrophie sénile symétrique des pariétaux;" 'Bull. Soc. anatomique,' 1876, p. 485; 1881, p. 414 et p. 72. "Contribution à l'étude de la pathogénie et de l'anatomie pathologique du céphalématome;" 'Revue mensuelle de Méd. et Chir.,' 1880.

act as direct anatomical causes ; but we must allude to an arrested development of the rachis, spina bifida, more or less incomplete ; to analogous defects of the skull and malformations of the head (asymmetry, microcephalism, &c.), and of the face (asymmetry, hemiatrophy, hare-lip, &c.) ; of the ear (congenital deafness, malformations of the concha, &c.) ; of the eye ; arrested development of limbs ; of the abdominal walls ; herniæ ; malformations of the genito-urinary organs, &c. All such developmental anomalies constitute the "stigmata" of the degenerative tendency, as they have been called.

A certain number of subjects, well formed at birth, are afflicted later on with deformities, such as strabismus, club-foot, &c., which have mostly succeeded to convulsions or spinal paralysis, &c., and of which the aspect often betrays the origin.

Anatomical lesions, however, are not the only characteristics observed in the subjects of nervous affections. On investigating their antecedents, one frequently finds traces of somatic or psychical troubles which constitute another group of "stigmata," viz. the *functional* premonitory troubles ("infantile preludes to neuropathies").

Among the functional somatic stigmata some are remarkable for the generalisation of their phenomena, whilst others are limited and remain unnoticed, or are looked upon with a certain degree of indifference. The importance ascribed to them varies also with the circumstances in which they have manifested themselves. Infantile convulsions, or eclampsia, are readily neglected in their prognostic significance, when occurring during dentition, after a blow, or mental shock, &c. The tetanus neonatorum and tetany often pass for accidental occurrences which will leave no trace. The same is the case for chorea, which we are accustomed to see treated as a complication of rheumatism, though it may also arise from traumatism or the puerperal state, conditions which, like rheumatism, do nothing but bring out the neuropathic predisposition. Localised spasms, even when accompanied by loss of consciousness, as for instance, the salaam convulsion or eclampsia nutans, are still more neglected ; and more so again the "tics," such as facial spasm, torticollis, spasmodic sneezing, nocturnal spasmodic cough, trismus (especially

nocturnal), asthma, laryngismus, troubles of articulation, &c. With reference to neuralgias, more especially sick headache, scarcely any stress is laid on them, though they are rare in children, and always constitute a sign of neuropathic predisposition. In addition to these phenomena, we must allude to nocturnal or diurnal incontinence of urine. The nocturnal kind has already been well studied by Trousseau, who has pointed out its significance. Diurnal incontinence has not yet attracted sufficient attention; it occurs in the waking state, and may surprise the child in the middle of his games. Micturition is irresistible, and may be either complete or incomplete; the urine is expelled by jerks, either single or repeated. This diurnal incontinence is often, but not always, associated with habits of masturbation.

Certain individuals have peculiar dysæsthesiæ, which may involve every sense; some cannot bear to be in the dark without being seized with an invincible terror. Hobbes is said to have been seized with a sort of delirium as soon as he was left without a light at night. Others cannot bear certain smells, even the most pleasant; the compositor Grétry, for instance, could not endure the smells of roses.

The psychical "stigmata" are neither less various nor less frequent. Neuropathic individuals have often presented from their childhood peculiar characteristics. Some are torpid, lazy and indolent, or work only by fits and starts. They dislike movement under any circumstances, keep away from noisy games and every bodily exercise. They are prone to depression, and seek to be alone; they often experience sensations of "malaise," of pain, of moral dysæsthesia. They have the "ennui" of themselves, they fear the future without cause: a spot on a copy-book, bad marks for their lessons, plunge them into a dumb anguish which may last for days. They are sombre, dreamy, taciturn, sulky, suspicious. Sometimes this state of psychical malaise presents acute recrudescences, states of anguish (*raptus melancholicus*), with temptations to crime or suicide. The act is then only a reflex phenomenon connected with the psychical anæsthesia or paræsthesia. The states of painful psychical depression with irritability occur chiefly at the onset of puberty. Others, on the contrary,

are excited, always on the move, cannot remain in one place, or preserve a correct attitude, and are subject to abrupt, unruly movements. They are equally mobile from a psychical point of view, their desires are as changing as their attitude; this inconstancy shows itself in their affections and their tastes; what pleased them yesterday does not please them to-day: what they like to-day will be disliked to-morrow. Hence they often manifest a tendency for change and roaming. Such variations are the more readily appreciated, that these subjects always manifest an exaggeration of emotional movements and feelings.

We must remark here, that we do not consider the emotions as essentially pathological phenomena. What is required to establish the morbid character of an emotional display is the disproportion between its cause and its intensity. When a decapitated frog is subjected to an excitation of its inferior limbs, the leg performs a movement of defence; if the excitation is stronger, both legs enter into play; beyond this point the four extremities are convulsed. These movements bear a certain proportion to the intensity of the excitation. If the animal has been previously strychnised, the least touch produces such a generalised and exaggerated reaction. We find in man an analogous state of things with reference to psychical reactions; in a normal condition these reactions are proportionate to the excitation. If a man utters a cry of terror when lightning strikes near him, the reaction is normal; but when a hysterical subject utters the same cry on perceiving a caterpillar at a distance, it is a morbid manifestation, showing the subject to be strychnised, so to speak, by a morbid heredity, or a defective evolution.

As a rule, individuals predisposed to nervous disorders are strangely timorous; many up to the age of twelve or fifteen cannot remain alone, nor sleep without a light, &c. Sometimes, notwithstanding this timidity, they display audacity for mischief. Some have a tendency to cruelty towards other children, or towards animals; they are young criminals. Although, as a rule, extraordinarily active, they are incapable of attention or application when the object is a useful one. They often manifest a premature development of the

sexual instinct which frequently takes an unnatural direction ; and excessive onanism still further exaggerates their depressed or emotional states, and brings into evidence the irritable weakness of the subject.

Finally, it is not rare for these individuals to show alternations of depression and excitation, which their friends attribute to variation of temper, and treat as unimportant, but which really constitute various phases of the same morbid state. These alternations are not unconnected with those attenuated forms of "*folies circulaires*," which M. Falret with much reason insists upon in his teaching. Such individuals are often seen to enter into a state of exasperation and convulsive passion, after a period of stupid resignation of variable duration. These fits of excitement break out sometimes with the suddenness of an epileptic attack ; it is a real change at sight. We know of two cases of this kind in which the violent fits of anger terminate in a deep sleep with loud snoring, which reminds one of an epileptic manifestation. Whether excited or depressed, these children usually show marked perversions either of an intellectual or of an emotional description, such as eccentricities and depraved feelings. Some are of a generally weakened intellect ; they learned to speak very late ; they were unable to acquire more than a most elementary instruction, learning slowly and forgetting quickly. Others, though of deficient intellect, manifest remarkable special aptitudes for music, for numbers, and the like ; "*partial geniuses*," as they have been called. These weak-minded individuals, besides a morbid obstinacy, manifest certain psychical plasticity, a peculiar suggestibility, which makes them liable to be incited to commit crimes beyond their power of conception. Thus we see them, according to the external circumstances, at times in a mood of kindness, at others extremely obnoxious. They are unable to bear a sorrow, and are easily frightened. Mental perversion and weakness are often associated, either form predominating ;¹ the tendency to pyromania, or dipsomania, or kleptomania, which is comparatively frequent among young subjects with neuropathic heredity, is rarely associated with a normally developed intellect.

¹ Kahlbaum, 'Allgem. Zeit.-chr. für Psychiatrie' x. 4, 5.

Notwithstanding their condition of intellectual inferiority, they are often endowed with a true psychical euphoria; they are satisfied with themselves, and this satisfaction, which they sometimes get those about them to share, enables them to succeed in certain easy vocations. To these feelings of self-contentment is often associated a domineering turn of mind. Such individuals are hard and overbearing to their inferiors, whilst they are obsequious and humble to their superiors.

In addition to these psychical peculiarities, which constitute, so to speak, an habitual state, other accidental phenomena are observed, such as nocturnal or diurnal terrors, often occurring in sudden fits. The diurnal terrors are frequently systematized. Some are frightened at the sight of certain animals or insects; thus, Meyerbeer could not stand the sight of a cat. Or the terrors may occur at various intervals, and involve a great number of objects. Again, they may be constant, and extend to a single order of ideas, such as the fear of death, which in children, usually so light-hearted, even in the course of grave illnesses, constitutes a truly morbid symptom. We need not insist upon the significance of nocturnal terrors.

It is only after a thorough inquiry into the existence of such organic or functional disorders, and into the neuropathic troubles of childhood, that one can determine the value of the exciting causes of nervous diseases. Otherwise there is a danger of attributing to traumatisms or to infectious diseases an importance which they do not possess in reality.

The mode of action of traumatisms in the production of nervous complaints is not always the same. (*a.*) Sometimes it brings into play an already specialised predisposition; as when the subject of hereditary neuropathic tendencies, who has had several attacks of chorea, suffers a relapse after a more or less localised shock; or in certain complications of surgical injuries, such as in the case of J. Hutchinson, where a patient died of tetanus whose father had died of the same complication. (*b.*) The traumatism may also localise a general predisposition, as when it calls forth a hysterical contracture. (*c.*) It may hasten the evolution of the disease, as in tabes dorsalis, and look as if it were the efficient cause of it.

It is certain that, apart from any hereditary or congenital

predisposition, certain traumatisms, and more particularly those of the skull, of which the action is more direct upon the nervous centres, create a predisposition which may be designated as accidental,¹ but which probably always implies an anatomical basis. When the individual who has thus been "neuropathised" by an accidental lesion, has recovered, he remains predisposed to neuropathic disorders; he has acquired a special pathological habit; his nervous system will preserve a peculiar excitability which may henceforth be called into play by various excitations. What we have just said of traumatisms we may repeat of moral shock, of acute diseases, of intoxications. Morel had not failed to observe the frequency of neuropathic complications after acute diseases. He quotes, for instance, a family of eight children, who, after typhoid fever, had every one cerebral symptoms, but where a hereditary tendency was at the root of these accidents. General diseases, like traumatisms, create neuropathic predisposition only if they have accidentally determined lesions of the nervous system. Under these circumstances the subject "inherits of himself," as Lasègue remarked.

When we take into account the hereditary tendencies and the early manifestations which betray a disturbance of evolution, we shall see that it is not the predisposition which it is necessary to prove, but it is rather the absence of this predisposition which is to be doubted in the history of nervous diseases.

¹ Lasègue, "Les Cérébraux;" 'Etudes Médicales,' 1884.

ON STAINING "IN TOTO" THE CENTRAL NERVOUS SYSTEM WITH WEIGERT'S HÆMATOXYLIN.

BY CHARLES E. BEEVOR, M.D., M.R.C.P.,

Assistant-Physician to the National Hospital for the Paralysed and Epileptic.

As I had the opportunity of working with Professor Weigert at Leipzig in 1882, and assisted him when he discovered his valuable method of staining the medullated fibres with acid-fuchsine, I have taken a great interest in his later discovery of staining with hæmatoxylin.

Ever since I saw his beautiful preparations shown at the International Congress at Copenhagen last summer, I have been trying to apply this last method to staining the central nervous system *in toto*, so that sections might be cut from the mass, and mounted at once without further treatment. The advantages to be gained by this method are of course very great, and a great amount of labour would be saved if a brain or spinal cord could be thus stained *in toto*.

My first successful preparations were made at the beginning of last October, and since that time I have continued working at this subject; as I have had a great many failures in trying to vary the method in different ways, I think that the means by which I obtained my first specimens will prove to be the most efficacious. This method consists in hardening the piece in methylated alcohol, then leaving it in potassic bichromate solution from one to four weeks, and putting it again into methylated alcohol for one to two days before using Weigert's hæmatoxylin.

The specimen on which I first worked was the whole brain of a marmoset—for which I have to thank Mr. Beddard, the prosector of the Zoological Gardens. By a mistake, this had been hardened in methylated alcohol for two or three weeks; it was then put into bichromate of potash solution, 3 per cent., for

another month; the brain was then cut frontally, *i.e.* vertically and laterally, into pieces about one-eighth inch thick, or less, washed in methylated alcohol for one or two days, and then put into Weigert's hæmatoxylin for four days, and on each day for three or four hours it was kept at the temperature of 40° to 50° C. The strength of the solution used was double that recommended by Professor Weigert, as I had found by a previous trial that it seemed to stain better than his original solution; I therefore used the following formula:

Hæmatoxylin	gr. xxx (2 grammes).
Absolute alcohol	min. 200 (14 gr.).
Water	oz. 4 (130 gr.).

The pieces of brain were then slightly washed in water and put into the solution of ferricyanide of potash (2½ parts), borax (2 parts), with water (100 parts); and this solution was changed till no more brown colour came from them; they were then washed in water.

The pieces were then passed through methylated alcohol, absolute alcohol, oil of cloves, oil of turpentine, and imbedded in paraffin, according to the method used at the Physiological Institute at Leipzig, and which I had learnt there under Dr. Gaule.

Sections were then cut, the paraffin removed by xylol, and mounted at once in Canada balsam.

A series of twenty-four of these sections mounted on two slides was shown at the Pathological Society during the past session; the first twelve showed all the naked-eye changes of structure, from the optic commissure to just in front of the pons, while the other twelve sections extended from the anterior part of the pons to about its middle. In these sections all the medullated fibres were very well stained.

I have since tried staining *in toto* specimens which have been hardened in bichromate of potash in the usual way, and afterwards put into methylated alcohol; but I have had so many failures with this method that I have been obliged to give it up. I have, however, stained the cord of a monkey *in toto* by hæmatoxylin, which had been hardened in bichromate of potash, 3 per cent., for two or three months; pieces were then put into methylated alcohol for a week, and then into

the strong solution of Weigert's hæmatoxylin for three days, and warmed for about three hours every day to 45° C. Sections cut from these pieces, after imbedding in paraffin, showed the whole white substance stained black, while the central grey matter had only its medullated fibres coloured.

I have further stained some of these pieces with picrocarmine *in toto*, after developing the Weigert process, and other pieces with eocene alcohol *in toto*, and have thereby brought out the cells of the grey matter and the neuroglia of the spinal cord in contrast to the darkly-coloured medullated fibres.

On the other hand, I have frequently failed to stain *in toto* with hæmatoxylin, after hardening with bichromate of potash in the usual way; and I have invariably failed when the pieces were put direct from bichromate solution into hæmatoxylin, without being first treated with methylated alcohol for a day or more. Where I have partially succeeded, the finest fibres of the grey matter were not properly stained, and often not at all.

With regard to the action of the bichromate solution; it does not seem necessary for the pieces to be hardened in this solution, as I have found on treating sections, cut from pieces hardened in alcohol, with bichromate solution for three to six days, and then immersing these sections for twenty-four hours in methylated alcohol, that they stain well with Weigert's method; and in this way I have re-stained sections cut from specimens which had been imperfectly stained *in toto*.

With regard to the new method with acetate of copper, published by Professor Weigert in the 'Fortschritte der Medicin,' April 15, 1885, I have as yet tried staining *in toto* with one specimen only, viz. a spinal cord from a case of locomotor ataxia. This had been hardened in bichromate of potash six weeks, and in methylated alcohol for about six months; pieces were put into the acetate of copper solution for five days, being warmed to 45° C. for three hours daily; then in methylated spirit for four days, changing the alcohol till no more green colour could be washed out, and in strong hæmatoxylin for four days, but without warming; the piece was then developed by ferricyanide of potash and sections cut from it. It was then seen that the whole of the white columns were

stained black, with the exception of the sclerosed portion ; but the part of the columns nearest to the central grey matter were not so deeply stained, and in places were hardly stained at all ; and the finest fibres of the grey matter were not stained well, and in parts not at all.

As these preparations have only just been made, I have not had time to try another specimen before writing this paper ; but I think if the preparation had been warmed to 45° C., or had been stained a few days longer, that the whole piece would have been penetrated, and I hope eventually to attain this.

I should remark that in the paper mentioned above, Prof. Weigert states that he has tried applying his method to staining pieces *in toto*, but that he has obtained only imperfect results.

From his article I presume, that he has made use of staining *in toto* with his original method only, and does not refer to his latter discovery with acetate of copper. With regard to the former method, I think that my sections made from the marmoset's brain show staining of the finest fibres generally, and certainly bring out the fine fibres in the corpus striatum and optic thalamus, while the larger masses of the internal capsule, optic tracts and corpus callosum are completely stained.

I regret that I have not, as yet, had time to try and perfect staining *in toto* with acetate of copper and Weigert's hæmatoxylin ; but in the one trial I made, nearly the whole of the white columns have been coloured black.

Clinical Cases.

TWO CASES OF SPINAL DISEASE ASSOCIATED WITH INSANITY: I. TABES DORSALIS; AND II. CHRONIC ATROPHIC SPINAL PARALYSIS.

BY WILLIAM DUDLEY, M.B. (LOND.),

Pathologist and Assistant Medical Officer, West Riding Asylum, Wakefield.

I.

A. B. æt. 54, single, surgeon, came under treatment in the West Riding Asylum, in March 1885.

Patient, who is an M.R.C.S. and L.S.A., entered the Army Medical Service, served in the Crimean War, and subsequently was a ship-surgeon for some months, visiting Australia. He again entered the army, and was in active duty on the West Coast of Africa for about three years.

Whilst there he had a sunstroke; he suffered also from "Gold Coast," or "bilious remittent fever," and contracted syphilis from a black woman. At this time he drank heavily.

He returned to England sixteen or seventeen years ago, but had not completely recovered from the effects of sunstroke, and was chiefly dependent on his sister for a livelihood. For a short time he carried on a low-class dispensing practice, until disabled by a paralytic seizure.

Patient has had three paralytic seizures. The first occurred seven years ago. "Whilst walking along the street he felt as if his left leg had been shot from under him. He gradually recovered the use of the limb. In another seizure the onset was sudden, and attended by loss of consciousness. He was dispensing medicine at the time. His sister coming into the Dispensary found him unconscious on the floor. Gradual recovery followed this attack."

He has become depressed and unmanageable, and keeps his bed for days together. Latterly he has become excited, deluded, and rambling in speech, disorderly, throwing anything which displeases him on the fire. He now suffers from incontinence of urine and fæces.

Family History.—His father and mother both died at an

advanced age. There is no history of any neurosis, phthisis or apoplexy. His father was intemperate.

Mental condition on Admission.—Patient is slightly excited, garrulous, affable; constantly calling for champagne and cigars. He answers questions readily and to the point; but his answers are given for the most part with little thought, and are unreliable. His memory is especially defective in regard to dates. Asked the date of to-day, he replies without hesitation, "25th Jan. 1885." He does not understand the nature of this place, although he has been told. He thinks it is a public-house, the "Bull and Mouth," and that he came here a week ago. When told again that it is a lunatic asylum, he endeavours to account for being here, by saying that he "went to Burnley a month ago on the 17th of March, with some friends; got drunk, and was sent here for safety." He declares that he had £550 in his pocket at the time, but that the money was taken from him.

He talks much in a very deluded, optimistic strain, *e.g.*, "I am a duke, and I will get the Queen to make you a duke to-morrow. She will do anything for me. She gives me £10,000 a year. I am going to Madeira on Monday in the Queen's yacht; will you come? I am going to buy an estate for £1000. I am going to be married to a duke's daughter. I will give you £10,000 when you marry my sister," &c. He takes up readily almost any grandiose suggestion that is made and adds to it. Thus when asked if he is still practising his profession, and if he has many patients, he replies that he is consulting physician to various European monarchs, enumerating half-a-dozen, and is in receipt of immense salaries from each. He offers £50 or £1000 indifferently to any one who will bring him a box of cigars. He is ready to scribble something perfectly illegible on any scrap of paper, and to give it as a cheque on his banker for any sum that any one desires. These "cheques" are filled up with the greatest expedition; the writing is merely a few wavy lines; he is in too great a hurry to write; it is such an unimportant matter to him. "Here, use my name, and they will give you the money."

He talks freely about the events of his earlier life, his memory for recent occurrences is more defective. With regard to the question of syphilis, he cannot state whether he had a true chancre or chancreoid; at the same time, or at some other, he had a bubo, which has left a scar in the groin. He treated the sore and bubo with nitric acid and lotio nigra, and took calomel, and iodide of potassium internally. When asked the dose of calomel, he replied, "a drachm twice or three times a day, that is the ordinary dose." He says he has had lightning pains in his legs, but not for many years past.

Physical Condition.—Height, 5 ft. 4 in. Weight, 124 lbs. Cranium broad; of good frontal development. Hair and beard white; crown of head bald. Limbs equally nourished.

Patellar reflexes are entirely absent. Plantar, cremasteric, abdominal, and epigastric are present. Sensibility of skin of lower extremities appears somewhat blunted. Contact sensations are fairly well localised. There are no areas of anæsthesia or of hyperæsthesia. When blindfolded, he is unable to distinguish whether he is standing upon a boarded floor, a rug, or a pillow. The floor, he says, feels soft like a carpet.

His gait is very distinctly ataxic. He walks slowly, with his legs widely separated, and the feet spreading; the steps are short; the legs are thrown clumsily forward; the feet are raised too high, and are brought down rather heavily. He uses his arms largely to balance himself during progression, and is unwilling to walk without assistance, or some object to grasp to steady himself. He keeps his eyes fixed on the ground about a yard in front of him, and if asked to walk while looking up, his difficulty is increased, and he frequently glances downwards, involuntarily, as it were. When asked to stop and turn round, he does so slowly and with difficulty. He can stand with much swaying to and fro for a short time while his feet are close together, but not at all with feet together and eyes closed. He is quite unable to stand upon one foot. He can move his limbs with a considerable degree of force in all directions. He has little control over his bladder and rectum. His writing, when he takes care, is quite legible, and not very shaky. His grasp is tolerably good; that of the left hand is decidedly weaker than that of the right. The tongue is large and flabby, *its right half is much smaller than the left*. It is protruded somewhat jerkily. It is almost free from tremor, and can be moved freely to either side, or curled up. He swallows without any difficulty. Facial expression is somewhat vacant; the facial muscles are usually flaccid, but they can be made to contract voluntarily.

Pupils are circular; very small; right slightly the larger; there are no iritic adhesions. There is complete irido-plegia both in response to light, and also with accommodation; nor is there any dilatation upon cutaneous irritation.

All ocular movements are well performed. There is no diplopia or nystagmus. Sight is good, except that he is somewhat presbyopic. There appears to be no contraction of the field of vision, and colours are distinguished perfectly. The pupils dilate fully under the influence of atropine, and the disc is quite healthy.

Hearing is slightly and equally dull in both ears. Taste is perfect. The sense of smell is blunted, or certainly not acute.

Heart sounds feeble; no murmur. Superficial arteries are free from evident atheroma. There is very slight arcus senilis.

Lungs are slightly emphysematous.

Tongue fairly clean, flabby. Teeth very deficient in number and quality. Appetite defective; bowels rather costive. Liver dulness normal.

Urine normal.

April.—He is now much less excited, and his answers are more reliable. His delusions are less prominent and less variable. He thinks now that he has been appointed Clinical Assistant here, with a salary of £200 a year, rising to £700; he is ready to commence duty as soon as his keys are given him. He says he is going to enter the Army Medical Service again at a salary of £500 a year. He can obtain a post as a matter of course, and thinks himself quite fit for active service. He is going to see the Director-General, the Queen, &c., with regard to it.

Since admission, there has been but little change in the patient's physical condition. He can walk a little better, and has much more control over his bladder and rectum. His pupils are not quite so small, still immovable; the left is a little the larger. Plantar reflex is well marked in the left foot, almost absent in the right.

He has given no indication of any subjective sensory impressions, nor has there been any gastric or other crisis. He takes food well, and his general health and condition have improved.

II.

R. P., æt. 61, married, formerly a labourer in a mill, was admitted into the West Riding Asylum on the 7th of March, 1885. The history obtained with him was imperfect and unreliable, but it was stated that he had been more or less insane for about two years prior to his admission; that his bodily health had been gradually failing for several years, and that for at least a year before admission he had been entirely confined to the house and had been supported by his children. It was also said that he had for many years been very intemperate, and that his wife and daughter were suffering from venereal disease contracted from him.

Family History.—He has four children, all of whom are said to be healthy. No history of insanity in the family, but his mother is said to have been paralytic for several years before her death.

Mental State.—On admission he was somewhat depressed and emotional, but manifested no dementia whatever; was perfectly

rational, and gave a coherent and detailed history of his past life. He stated that about nine years ago, while working in a flour mill, he one day accidentally put his left leg through a trap-opening, the door of which had been carelessly left open, and that his body fell across and struck the edge of the trap. He did not feel much hurt at the time, and was able to go about his work as usual for about a week after the occurrence of the accident. At the end of that time, however, owing to gradual loss of power in the left leg, he was led to seek medical advice, and went into the Leeds Infirmary. After he had been there three days he became completely paralysed in both lower limbs; but after a course of galvanism, lasting for three months, he was able to walk with the aid of sticks. He stated that he never recovered half his former power in the affected limbs, but that about nine months after the accident he had recovered sufficiently to be able to go back to his work at the mill, at which he continued for about two years and three months, when he met with another accident. While doing something in connection with the working of the machinery, a weight of about nine stones fell upon his left hand, and he lay for several hours in that situation before he was discovered and released. On account of loss of power in the arm, he was taken next day to the Infirmary; but, in spite of treatment, the arm gradually wasted. He noticed that the right arm began to lose power about nine or twelve months ago, and it has gradually got worse.

Visual acuity has gradually diminished during the past three or four years, and his voice has altered in character during the past twelve months or so.

Physical Condition.—Height, 5 ft. 1 in.; weight, 94 lbs. Cranium rather small, dolichocephalic. There is an old scar about 1 inch in length running antero-posteriorly, situated at the superior angle of the occipital bone. Beneath this scar is a distinct depression as if from fracture. Hair is grey and thin, and the greater part of the vertex is bald. Irides greyish brown. Pupils, circular; diameter of right, $5\frac{1}{4}$ mm.; of left, $4\frac{1}{2}$ mm.; do not react to diffused light, nor to a concentrated beam, nor consensually: contract very slightly with accommodation: do not react to cutaneous stimulation. No reflexes can be obtained in the lower extremities.

Tongue is protruded straight. It is free from tremor, and its movements are fairly well co-ordinated. The lips and facial muscles are steady.

All the limbs are much wasted, the atrophy being more marked in the upper than in the lower extremities, and the left arm and leg are distinctly smaller than the right.

MEASUREMENTS

Right leg	..	5 in. below knee, $9\frac{1}{4}$ in.—Left, $8\frac{1}{2}$ in.
Right thigh	..	6 in. above knee, $13\frac{3}{4}$ in.—Left, $13\frac{1}{2}$ in.
Right forearm	..	5 in. below elbow, $5\frac{1}{2}$ in.—Left, $5\frac{3}{4}$ in.
Right arm	..	4 in. above elbow, $7\frac{1}{4}$ in.—Left, $6\frac{1}{2}$ in.

There are no contractures of the limbs, except in the fingers to a slight degree. The muscles generally are very flabby and extremely wasted. Idio-muscular contraction is scarcely perceptible in any limb. The left leg has a notably incurved aspect, apparently due to wasting of the peronei muscles. This appearance is absent on the right side.

The upper extremities are extremely wasted. The forearms and hands are literally almost nothing but skin and bone. Many of the muscles appear to have disappeared entirely. The forearms are flattened upon the dorsal and palmar aspects. The thenar and hypothenar eminences have disappeared, and there are deep depressions between the metacarpal bones. Both wrists are dropped. The ungual phalanges are incurved so as to give the hand a clawed appearance, and this is more marked in the right than in the left. The upper arm and shoulder-muscles, especially of the left side, are also extremely wasted, as are likewise, but to a less degree, those of the back.

The lower extremities are more wasted proportionally below the knee than above; and the anterior and external muscles of the leg have suffered more than those of the calf.

The patient is unable to approximate the thumb and fingers. He completely fails to grasp with the left hand, and with great effort succeeds only in doing so very feebly with the right. He can neither pronate nor supinate the hands, nor can he flex or extend the wrists. He can perform very limited and feeble movements at the elbow and shoulder, and somewhat better on the right than on the left side.

As regards the electric reactions of upper limbs, serial alterations present themselves in both rhomboidei and left pectoralis major: the anodic closing contraction had nearly overtaken the kathodic closing contraction in the right brachialis anticus and left triceps: whilst no faradic or galvanic reaction could be obtained with the supinators of the forearm, the extensors or flexors of the hand and wrist, the intrinsic muscles of the hand and the right pectoralis major.

The reactions may be tabulated thus:—

FARADISM.		GALVANISM.
Deltoid	(right) Minimum at 9.	KSZ > ASz.
	(left) 0	KSZ > ASz.
Rhomboid	(right) 0	ASZ > KSz.
	(left) 0	ASZ > KSz.

FARADISM.			GALVANISM.	
Biceps	(right)	0	KSZ	$> ASz + AOZ.$
	(left)	0	KSZ	$> ASz + AOZ.$
Brachialis A.	(right)	0	KSZ	$> ASz.$
	(left)	0	KSZ	$> ASz.$
Supinator longus	(right)	fair with weakest current	KS—AS.	
	(left)	0 with strongest	KS—AS.	
Extensors of Hand	(right)	} 0	KS—AS.	
(extrinsic)	(left)			
Flexors of do.	(right)	0	KSz	$> ASz.$
	(left)	0	KS—AS—	
Serratus Magnus	(both)	0	KS—AS—	
Pectoralis Major	(right)	0	KS—AS—	
	(left)	0	ASZ—KS—	
Triceps	(right)	minimum at 7	KSZ	$> ASz.$
	(left)	do. at 6	KSG	$> ASz.$
Intrinsics of Hand		0	KS—AS—	

He is quite unable to stand. When asked to do so he struggles wildly. If assisted into a standing position, he throws his arms and legs about in a disorderly fashion. His lower limbs double at the knees as soon as any weight is allowed upon them. When lying on the bed, with the legs extended, he can succeed in partially drawing them up, but can exert very little force if resisted. He can cross his legs, but cannot place the heel of one foot upon the toes of the other.

There appears to be a great deal of muscular starting, accompanied by distressing pain. He states that he has been subject for some time to excruciating pains in the limbs and body, and which he describes as like flashes of lightning.

Sensibility to pain is retained in the skin of the face and scalp, and to some extent in the neck, although in the latter situation it appears to be blunted.

At tip of right forefinger, he distinguishes two points, 1 centimetre apart.

At tip of left forefinger, at 2 centimetres apart.

In centre of right palm, at 3 centimetres.

In centre of left palm, at 4 centimetres.

On front of forearms he cannot distinguish two points 10 centimetres apart.

Sensibility to tactile impressions and to pain is considerably diminished in the lower extremities, and sensation is much delayed.

The integument in several regions is dry and scaly. On the front and outer aspects of the legs this condition is sufficiently marked to merit the term "ichthyosis."

Thoracic and abdominal viscera appear fairly healthy.

The patient has little control over his bladder and rectum.

At all times he has but little sensation during the passage of evacuations, and is frequently quite unconscious of the fact.

The senses of taste and of hearing are fairly acute, that of smell is abolished.

June 13th.—Since admission there has been no change in the patient's condition worthy of record.

CASE OF CEREBRAL ABSCESS.

BY ARTHUR E. W. FOX, M.B., F.R.C.P. (EDIN.),

Physician to the Royal United Hospital, Bath.

ADELAIDE B., aged 25 years, a servant, was admitted into the Royal United Hospital under my care on January 28th, 1885. For the following notes of her case I am indebted to our House Physician, Mr. Edward Cave.

January 29th.—The patient states that she enjoyed excellent health until a fortnight ago, when she cut the middle finger of her left hand; it bled freely, and she was much frightened. In the evening of the same day, she lost the use of three fingers on the same side, viz. the middle, ring, and little fingers. There was no abnormal sensation; simply loss of power in the extensors of these fingers, and this continued without change until four days ago, when the loss of power extended to the rest of the hand and forearm, and she has been unable to do anything with it since. She applied to the Hospital yesterday morning, but feeling very faint on her way home, was brought back, and was at once admitted as a patient into Victoria Ward. At the time of her admission it was noticed by Mr. Roberts, our late house-surgeon, that there was slight convulsive twitching of her left face and left leg.

In appearance she is a nervous-looking girl, very well nourished, with plenty of colour in her cheeks, in fact looks very well; is of a clear brunette type.

She has never been laid up by any illness; never had any form of paralysis; never aphonic; no present or past otorrhea; no history of any blow on head; never had any kind of fit.

There is slight choreic movement of the right upper extremity; no twitching of the face or legs; pupils equal, moderately dilated; contract readily to light; no paralysis of any of the facial or ocular muscles; none of the tongue, palate or larynx. The left arm and hand are paralysed, and the hand is slightly blue and congested as compared with its fellow, and a little colder to the touch. Although the movements of the left upper arm are impaired, the paralysis is most marked in the forearm and hand. She can move the limb, as a whole, freely in any direction, but she cannot flex, extend, supinate, or pronate the forearm or hand, nor can she move any of the fingers; there is nothing characteristic in the shape of the

hand, the fingers are very slightly flexed, and the thumb half opposed to the index finger. There is no loss of sensation. She can localise a touch accurately, but a moderately severe pin-prick produces no expression of pain. All the muscles contract readily to the faradic current. She winces a good deal when pressure is made over the upper dorsal spines. She has no headache, is not giddy, can walk quite well. There are no signs of disease in the chest or abdomen. Appetite always capricious, and has been indifferent lately. No nausea or vomiting. Tongue coated with yellowish moist fur; bowels well opened yesterday. Menstruation regular. Urine sp. gr. 1030, with no albumen or sugar. Temperature 98·4, R. 20, P. 90.

January 30th and 31st.—Much the same, no headache, vomiting or pyrexia. On *February 1st* felt rather sick, but did not vomit. *February 7th*, complained of a little frontal headache. Continued much the same until *February 11th*, when for the first time, she vomited her breakfast. No headache, no pyrexia; if anything, can move her left arm, as a whole, more freely, but the forearm and fingers are still absolutely paralysed.

On *February 12th and 13th* she vomited several times; vomit, yellow and bilious. On *February 14th* complained again of frontal headache, not localised, nor worse on one side than the other; no tenderness of scalp; nor pain complained of on moderately hard percussion over the vertex. No other alteration in her state.

On *February 15th* did not sleep all night; was very restless and tried to get out of bed. No actual delirium. Complained much of her head. Has not vomited since yesterday morning; bowels confined, abdomen much retracted. Pulse 54, regular, and of moderate volume. She has not spoken since 6 A.M. and this morning is heavy and torpid. The left arm is absolutely paralysed, but she moves her right limbs and, to a less extent, the left leg in an irregular restless way. Does not protrude her tongue when told. Facial and ocular muscles act normally. Knee-jerks normal; the plantar reflex slight, but obtainable. Both optic discs appear to be somewhat too red, and the veins rather large; no swelling of either disc, no hæmorrhages. There is no expression of pain on smart percussion of the head, but she moans and struggles when deep pressure is made over the cervical and dorsal spines. There is no retraction of head. She became perfectly comatose, and died about 6 P.M. the same evening.

Post-mortem, twenty-two hours after death.

Heart, lungs, liver, stomach, spleen and kidneys healthy. Ovaries and fallopian tubes normal. First molar tooth in upper jaw on left side is slightly carious, and the adjacent

bicuspid is reduced to a stump; both were extracted, no suppuration at the root of either. Nothing amiss in either Antrum of Highmore. No otitis; on removing the calvarium, dura mater appeared normal; was not adherent to the bones. There was no disease of the bones or evidence of any injury to the skull. On reflecting the dura mater, a prominent, convex, and fluctuating swelling was apparent on the surface of the right hemisphere, chiefly in front of the superior end of the fissure of Rolando, and extending down along the ascending frontal convolution. An incision was made into the swelling, and about $\frac{1}{2}$ an ounce of pus evacuated. There was nothing amiss at the base. No meningitis. There was an average amount of subarachnoid fluid. The brain was placed in spirit



FIG. 1.—LATERAL VIEW OF HEMISPHERE. The white patch indicates position of abscess; the shaded patch, that of the softened brain matter.

and water to harden. Examined, after hardening in spirit, the abscess was found to be as big as a pigeon's egg, coming to the surface and so involving the grey matter, at the anterior margin of the ascending frontal convolution, 2 inches from the longitudinal fissure. (See figs. 2, 3, p. 254.) It extended obliquely, inwards and downwards, for $1\frac{1}{2}$ inch towards the corpus callosum, while anteriorly it involved the posterior part of the superior frontal convolution. There was some reddish softening of the posterior portion of the second frontal. The abscess cavity was lined by a thick pyogenic membrane. The rest of the brain appeared perfectly normal.

Remarks.—This case is of interest in its insidious origin and comparatively latent course. The origin of the abscess is very obscure; there being no ascertainable history of injury to her

head, neither had she any bone disease, nor, as far as we could find, any pressing focus of suppuration. Lebert would probably regard the case as one of idiopathic cerebral abscess; but, in the face of the opinions expressed upon this subject by Gull, Sutton, Wilks and others, I confess that, though we took a great deal of trouble in our search, I consider that we must have overlooked the primary cause. Amongst other curious features in this case, are the small amount of headache and vomiting, and the complete absence of pyrexia from first to last. She never complained of headache, when questioned

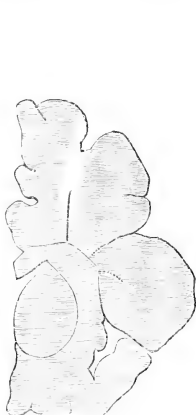


FIG. 2.—FRONTAL SECTION.

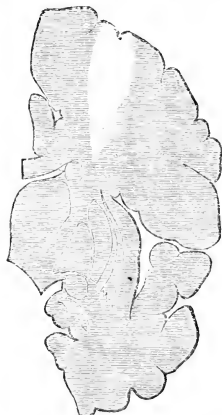


FIG. 3.—PEDICULO-FRONTAL SECTION.

Showing position of abscess, and of softened region.

upon the subject, until February 7th, eight days before her death, and then it was never strictly localised, was of an intermittent character, and never severe until the last twenty-four hours of her life. The point of paramount importance in connection with the case appears to me to be the bearing it has upon localisation. The abscess involved, pretty accurately, the centre which Ferrier has laid down for supination of the hand, flexion of the forearm and extension of the arm and hand, the very parts paralysed in this girl's case. The fact that the muscles first affected were the extensors of the middle, ring, and little fingers of her left hand, corroborates Ferrier's statement,¹ that "a generally enfeebling cause will show itself first prominently in the extensors."

¹ 'BRAIN,' Part XV., page 311.

NERVE SUTURE; STRANGULATION AT POINT OF JUNCTION; OPERATION. RAPID RECOVERY OF SENSATION AND MOTION.

BY WALTER PYE, F.R.C.S.,

Surgeon to St. Mary's Hospital.

H. B., 25, a cellarman and bottle-washer, was admitted into St. Mary's Hospital, March 12th, 1885, with the symptoms of paralysis of the left ulnar nerve.

History.—He stated that at the end of 1883 he sustained a deep cut along the inner border of the left hand, through a bottle breaking as he was washing it. He went to the Royal Free Hospital, an anæsthetic was given, and some fragments of glass removed. The wound was dressed, and healed up directly. There remained, however, shooting-pains in the first and second fingers, with anæsthesia of the fourth and the ulnar side of the third fingers. He tried to resume his work; but a fortnight afterwards the wound broke out again, and he returned to the hospital.

Under an anæsthetic, Mr. Rose then opened up the wound by an incision made over the position of the ulnar nerve, at the wrist and bottom of the palm. The nerve-trunk was found to be divided, and was united end to end. The wound healed with a moderate amount of suppuration, but without further trouble. From that time until his admission into St. Mary's, there was no return of sensation in the fingers, while the scars, both of the original wound and of the surgical cut, became very acutely hyperæsthetic. There was also steadily increasing wasting of the palm of the hand, which got more and more clumsy and weak, so that he could in no way follow his trade. He finally came to Mr. Pye's out-patient room at St. Mary's, and was admitted into the wards.

Condition on Admission.—A healthy man, but poorly nourished.

The left forearm is marked in several places by the scars of cuts said to have been inflicted by fragments of bottles broken by him whilst washing them; the muscles of the forearm appear to be all equally developed. Along the course of the ulnar nerve, at the junction of the wrist and palm, there is a longitudinal scar crossed obliquely in the palm by another

(the oblique scar was that of the original wound; the long one, of the operation-wound). The former is much indurated, and in the centre is scabbed over only, but is firmly healed in the rest of its extent. The scab is exactly at the junction of the wrist and the palm, and here especially, but to a less degree over all the scar, there is an apparently acute hyperæsthesia, with reflex spasm of the flexors of the wrist produced upon the slightest touch.

The patient states that he cannot feel at all with the little finger and with the ulnar side of the ring-finger; but examination shows that, although the sensation is extremely impaired and altered, it is not quite abolished, so that he is conscious of a deep pin-prick, or a firm squeeze, although neither gives him pain; tactile sensation appears to be quite absent.

The hand is generally ill-nourished, probably from disuse, but the spaces between the metacarpal bones are especially wasted, and particularly the interval between the thumb and the first finger, this last being plainly due chiefly to atrophy of the adductor pollicis. The movements of the digits are clumsy, but can be performed, with the exception of complete adduction of the thumb and complete extension of the fingers.

Sensation in the palm, with the exception of the parts mentioned above as being hyperæsthetic, seems to be natural.

March 21st.—The patient was anæsthetised, and an incision was made following the line of the former one, being therefore in the line of the ulnar trunk. The nerve was exposed in the healthy tissues of the upper part of the wound, and traced downwards, being carefully dissected out from the dense cicatricial tissue in which it lay imbedded. It was in this way followed right through the scar, and down to its division into its superficial and deep branches in the hand.

These branches presented a normal appearance, as did the upper part of the trunk where it was first exposed, but between these two points it was obviously strangulated by the fibrous bands of the scar tissue; these ran through it and around it, the tightness of some of the circular constrictions being evidenced by the nerve swelling out on either side of the bands.

At the place which was obviously where the nerve had been originally divided and subsequently reunited, there was a well-marked bulbous enlargement, of about the size of, and presenting an appearance very similar to, the Gasserian ganglion.

As much of the fibrous infiltration and strangulating investment of the nerve-trunk as it was possible to dissect off without injuring the nerve-fibres was then carefully removed with the

scalpel, and the small nerve bundles separated one from another; the wound was closed, a short and very fine drainage-tube inserted, and the hand and forearm splinted lightly.

With regard to the wound, all that need be said is that its original dressing was never disturbed; and that it healed straightway without a drop of pus.

The following is the record of the process of return of sensation:—

March 25th.—Until to-day patient has noticed no difference in the fingers from their condition previous to the operation, but he now (four days after) thinks the third finger feels more natural; no change in the little finger.

March 26th.—The third finger is distinctly less numb, in his opinion, and the little finger is beginning to improve.

March 29th.—Hand removed from splint; the wound is healed. The improvement of sensation has continued, and he says that he feels as if he had more use in the hand. There is no perceptible alteration in its wasted appearance. There is no hyperæsthesia in the region of the operation-wounds, where it was very marked before.

April 9th.—The sensibility of the parts supplied by the ulnar nerve now seems to be quite normal and as good as upon the other side of the body; he is confident as to increased usefulness of the hand; the wasting is, if anything, less.

April 11th.—Discharged, to recommence his work. He was afterwards seen frequently in the O. P. room at the hospital. The sensibility of the ulnar region is up to the present date (June 28th) permanently restored, and the weakness and atrophy of the affected muscles has steadily diminished, so that he now reckons that his left hand is about as good as his right one.

Remarks.—The whole interest of this case lies in the very rapid recovery of function shown by this nerve-trunk, after a suspension of its activity for a period of sixteen months after it had been divided and spliced together. This functional activity was, however, held in abeyance only, not abolished, and this was evidenced by the existence of some sensation, although it was only a very little, before the second operation. The atrophy of the muscles of the palm which are supplied by the ulnar was sufficient proof of the genuine nature of the case, and it is worthy of notice that the nerve-trunk below as well as above the seat of strangulation did not show, to the naked eye, any sign of impaired nutrition.

The recovery was, however, in any case singularly rapid and very complete, and the case would seem to encourage us to cut down upon and examine the condition of nerve-trunks, when

an operation for the reunion of their divided ends has not been followed by restoration of function, more frequently than is at present the practice.

It is well also to note the great importance of avoiding, in these operations, suppuration and the delayed union which it entails. This point has been already insisted on by writers on the subject of nerve suture.¹

¹ Vide Mr. Herbert Page's paper. 'Brit. Med. Journ.' vol. ii., 1880, p. 347.

Reviews and Notices of Books.

Les Maladies de la Personnalité. Par Th. RIBOT, Directeur de la 'Revue Philosophique.' 1 vol. 18mo., pp. 174. Paris: Félix Alcan.

THE leading idea in this very excellent and important work is the expansion of the "sixth sense." In all metaphysical works the existence of sensations over and above those which arrive by the five senses is mentioned, but until this work of M. Ribot, a bare mention is all the notice that these so-called "organic" sensations have received; and the extremely important rôle that they play in mental life has escaped appreciation. It has long been recognised that the visceral or organic sensations were a factor in the totality of consciousness, but the precise part played by them, and the very great importance of their share in the constitution of the conscious *ego*, have been worked out by M. Ribot in a way that has never before been attempted; and that marks a real and considerable step in the progress of the science of Mind.

When Psychologists of the physiological school have investigated the material basis of Mind, they have understood by Mind definite states of consciousness—percepts, concepts, memories, emotions. They have investigated the conditions of *that which is known* apart from and without regard to the conditions of *that which knows*. Discussions on the nature of the *ego* have been left to the introspective school, and have not been illuminated by the light which physiological investigation has thrown on the other departments of mental science. M. Ribot sets himself, in this volume, the task of applying in this most obscure region the same luminous principles which have enabled such great advances to be made in other directions, with the result that we are now able to say, that "the people that walked in darkness have seen a great light, and they that dwell in the land of thick darkness¹ upon them hath the light shined."

Although professedly treating of disorders of the personality, and marked off into chapters treating respectively of organic, affective, and intellectual disorders, yet the work is essentially physiological rather than pathological, and the facts of pathology are used mainly for the purpose of illustrating and enforcing the author's doctrine of the material basis of the conscious subject.

In order to appreciate the real concrete personality, and not an abstraction that is mistaken for it, we must not, says M. Ribot, shut ourselves up in our own consciousness with closed eyes and

¹ Revised Version.

obstinately question it; on the contrary, we must open our eyes and look about us. The child, the peasant, the artisan, millions of people who are going about their business, who have never heard of Fichte and Maine de Biran, who have never read an essay on the *ego* and the non-*ego*, have nevertheless a perfectly distinct idea of their own personality. It is necessary to account for this conviction of the multitude as well as for the uncertainty of the philosopher, and the explanation is to be found in the introspective view of the one and the practical experience of the other.

The objections so commonly made by a class of superficial thinkers against all introspective study are really valid when applied to the introspective study of the personality, and it is to this limited portion alone of the introspective method that these objections are applicable. By introspective observation we can observe states of consciousness, and these only, and we cannot hope to discover by analysis of these states a synthetic whole such as we believe the personality to be.¹ Hence, metaphysicians have considered states of consciousness mere accessories, and they have postulated the existence of some mysterious substratum which eludes observation, as the canvas on which the scenery of consciousness is painted, or rather as the sheet on which its dissolving views are displayed. It is this hypothetical substratum which, under the name of unity, or identity, or continuity, is considered the true *ego*. It is clear, however, that this principle of individuality is nothing but an abstraction—a verbal explanation which we have been content to accept for want of clear insight.

According to M. Ribot, the conscious *ego* is the subjective expression of the objective unity of the organism. Objectively and subjectively the characteristic feature of the personality is the continuity, the permanence, which we call identity. The conscious identity is nothing but the internal manifestation of the external identity which we witness both in ourselves and in others. When fully expanded, this expression has a very clear and definite meaning, and captivates more and more the judgment of the reader.

It is an axiom of physiological psychology that all consciousness is an accompaniment of nervous action. Without nervous action there can be no consciousness; but, on the other hand, there is an immense field of nervous action that has no conscious accompaniment. Nevertheless, there is no definite and no constant division between the portion of the nervous system whose activity is accompanied by consciousness, and the portion whose activity is not so accompanied. Ordinarily, that is to say, in normal waking life, the most vivid consciousness accompanies action of some part of the highest nervous centres, and there is a gradual shading off of consciousness—a gradation of tones and sub-tones corresponding with activity of centres similarly grading down towards the

¹ The true *ego* is that which feels, thinks and acts without itself coming into consciousness. It is by its very nature the subject, it is *subjectissimus*. To become objectified in consciousness it must undergo a transformation which must change its very nature.

lowest. It is to the highest centres that are mainly carried those impressions that we receive from the outer world by our sense organs. It is in these centres that are mainly registered the impressions of sight, of hearing, of taste, &c., and it is to these impressions that the attention of psychologists, and especially those of the introspective school, has been almost wholly devoted. But in addition to impressions of this class, there is an enormous aggregate of impressions arising from within the organism which are continuously flowing towards the superior nervous system, and which must be taken into account before our concept of the substratum of consciousness can be complete. Nerve currents starting from the whole of the interior of the organism—from the mucous and serous surfaces; from the viscera, the muscles, the bones, the articulations; from the arteries and even from the *nervi nervorum*—are continually flowing upward to the brain; their waves are continually breaking on the shore of the great central nervous territory—breaking not always on the same line, but nearer to or farther from the highest centres, according as the tide of nutrition ebbs and flows in the organism at large. Just as our consciousness of the world around us—object-consciousness—depends on the impressions brought by the nerves of sense to the highest centres, so our consciousness of our own personality—subject-consciousness—depends on the impressions brought by the visceral nerves to centres chiefly below the highest. Remark how the subject-consciousness corresponds in all respects with this aggregate of impressions pouring in from all parts of the organism. Note first, that while all states of consciousness by which we know the environment are intermittent, the consciousness of being—the personality—persists throughout. The continuous, ever-present, subject-consciousness corresponds with the continuous, ever-present, wash of the waves of the *cenæsthesis*. The intense nerve-currents set up by the impact of disturbances from without upon the organism, correspond with the intense states of consciousness that arise from them. Lights, sounds, tastes, smells, touches, are of various degrees of intensity, but all are of high intensity compared with the deep ground-swell of feeling that arises from the interior of the organism itself, and maintains a consciousness of small intensity, it is true, but ever present, of the continuity of our existence. Conversely, the state of consciousness aroused by even the greatest of the external disturbances is of small volume compared with the consciousness, faint though it be, but voluminous and all pervading, of continuous identity of the subject.

Even in sleep, when the impressions from the outer world gain no entrance, we continue in dreams to be conscious of ourselves; and why this subject-consciousness remains will be evident, when we remember that the impressions on which it depends are distributed largely in the lower centres, and that the diminution and arrest of activity which constitutes sleep begins in the highest centres and spreads gradually downward.

If this be the constitution of the physical substratum of the

subject-consciousness, then it is evident that the *ego* is not a homogeneous unit, as the metaphysical doctrine implies, but an extremely complex whole. It is not an "essence," but a co-ordination. Its physical substratum being in nerve-centres which represent every portion of the organism, it is evident that an alteration in any portion of the organism must be represented by a corresponding alteration in the nerve-centres—must involve a corresponding alteration in the *ego*. Hence as a body grows old the individual feels aged; not by deliberately counting up the years that he has lived, but by the mirrored representation in consciousness of the changes that the body has undergone. Hence as the body changes by the addition or subtraction of functions, as at puberty and the menopause, so the individual changes by the addition or subtraction of faculties. Hence, a high state of vigour in the nutrition of the body is mirrored in buoyancy of spirits; and imperfect tissue-changes, in mental depression. It must not be forgotten that the brain itself is a viscus, and as such contributes its share to the molecular turmoil of the *cœnæsthesis*. On the existence of a high degree of tension in the nervous elements depends the sense of energy, of capacity to act strenuously, which is so important an element to both the existence and the feeling of well-being; and on the capacity to store large amounts of force in the nerve elements depends the quality on which more than all others depends success in life—the quality that is known as "staying-power."

The total consciousness that we have at any moment is a compound of the subject-consciousness, and the object-consciousness. Hence, if the subject-consciousness changes, our whole consciousness will not be the same even although the object-consciousness remains unchanged. Hence it is that we see things, as we say, in a different light. They are the same, and yet they are changed—to us. "After ten years of absence, an object, a monument, is seen the same, but it is not *felt* the same." There is a difference, sensible but intangible. There is a curious feeling of identity commingled with novelty, due to the commingling of an identical object-consciousness with a new subject-consciousness. "'Tis we, 'tis ours, are changed, not they."

It is unnecessary to follow M. Ribot through the various disorders of the personality of which he treats. They are considered more for the light that they throw on the physiology of the subject than for their intrinsic importance, great as this undoubtedly is. It is sufficient to say that no book treating of this subject approaches the value of M. Ribot's admirable treatise, and no student of psychology can afford to neglect to make himself master of it.

Gehirn und Auge. By Dr. LUDWIG MAUTHNER. 1 vol. 8vo., pp. 255, with 10 woodcuts. Wiesbaden; J. F. Bergmann, 1881.

THIS work, which forms part of the first volume of Professor Mauthner's 'Lectures on Ophthalmology,' has already proved its position as an authoritative account of the subject it treats. Professor Mauthner's method is that of historical development. He nowhere begins his account of a subject with a statement of the present opinion regarding it, but traces the formation of opinion through its various phases, showing how successive fallacies have been eliminated, and where we are still in uncertainty. This method has its advantages and disadvantages: but considering the difficulties of the subject, and the fact that to a certain extent each case of central nervous disease with ocular symptoms has to be thought out in a way very similar to this historical development, we consider the method has much to commend it.

Professor Mauthner, after a short account of the development of the optic nerve and retina, commences with much-needed definitions of the varieties of hemiopia, hemianopsia, or, as he terms it, hemianopia. It is much to be regretted that there is so much confusion on this head. Are hemiopia and hemianopsia synonymous terms? Do they refer to the retina, or to the field of vision? Does lateral hemianopsia mean defect of the temporal halves of both fields of vision, or of homonymous halves? Dr. Ross in his work on the Nervous System makes hemiopia refer to the retina, *i.e.* to the blind half of the retina; hemianopsia to the field of vision, *i.e.* to the defective half—a distinction certain not to be carried out from the very structure of these words, and still more certain to cause confusion. It seems that it would be better if the term hemiopia were dropped entirely. Hemianopsia, from the analogy of hemiplegia, hemianæsthesia, might be taken as referring to the retina, but general consensus makes it refer to the field of vision. Hemianopia homonyma dextra or sinistra means, then, according to Professor Mauthner, the loss respectively of the right or of the left halves of both fields of vision; hemianopia heteronyma lateralis or temporalis, of the temporal halves of both fields, and hemianopia heteronyma medialis or nasalis, of the nasal halves. The meaning of hemianopia homonyma superior or inferior, and of hemianopia heteronyma supero-inferior will be readily intelligible. Hemianopia should not be used as meaning both sides, unless the term homonymous or heteronymous is added. Professor Mauthner gives an account of each of the varieties, with an analysis of numerous clinical records. Under this head he discusses exhaustively the arrangement of the fibres in the optic nerves, chiasma, and optic tracts, with full reference not only to the pathological lesions of these parts, but also to development and physiological experiment. His own work on Amaurosis in 1872 marked an era in this inquiry. Dr. Mauthner's explanation of the ophthalmo-

scopic appearances in an old hemianopia, and of the various complaints of hemianopic patients, are particularly clear and interesting.

Taking up next the more difficult subject of the course of the optic nerve-fibres in the brain, Dr. Mauthner discusses first Charcot's scheme of a supplementary decussation in the anterior corpora quadrigemina. This, principally on the ground of Muok's experiments on the visual centre, he rejects, showing that Charcot's cases of hysterical hemianæsthesia, on which largely his plan was based, in reality do not support the scheme, but, so far as they go, strengthen the belief that each optic tract ends entirely in its own hemisphere, viz. in the occipital lobe. This latter view is supported and discussed along with an interesting section on "Seelenblindheit." We could have wished that the nervous symptoms accompanying hemianopia had been more fully discussed. This part of the work is meagre and unsatisfactory, and we doubt not that, with increased material, Dr. Mauthner will in his next issue make substantial additions to this important portion. A full and clear account is given of the various appearances in neuritis and neuro-retinitis associated with central disease, and of the various theories as to their causation. The work finishes with a somewhat fragmentary account of the other ocular symptoms in diseases of the nervous system.

JAMES ANDERSON, M.D.

Ophthalmiatische Beiträge zur Diagnostik der Gehirnkrankheiten. By Dr. HERMANN WILBRAND. 1 vol. 8vo., pp. 100, with coloured plate. Wiesbaden: J. F. Bergmann, 1884.

THE anatomy and functions of the retinae, optic nerves, commissure and tracts are now, we may say, matters of certainty. We know the effect of lesions of each of these parts, and these effects are in agreement with known anatomical arrangement. One point alone, perhaps, is inferred independently of known structure—namely, the bilateral central connection of each macula. This assumption seems the most natural explanation of the escape of macular visions in both eyes when the lesion of the visual tract lies posterior to the optic commissure. At the same time, direct intracerebral commissural connection of optic centres cannot be regarded as excluded. As the optic tracts, however, disappear from view through the corpora geniculata, our certainty ceases both anatomically and physiologically, although we are not absolutely without light. The clue is almost wholly physiological and pathological, and seeing that decisive cases must necessarily be few, and will also seldom be observed with the requisite knowledge and accuracy, the progress of certainty in this region will necessarily be slow. Dr. Wilbrand, in his work on Hemianopsia and its relation to the localisation of Cerebral Diseases, has

already gathered up many of the results derivable from pathological lesion, and the present work may be regarded as a continuation and extension of the former.

Dividing the visual sense into light-perception, form-perception, and colour-perception, Dr. Wilbrand devotes Chapter I. of his work to the statement of various propositions as to the position and relation of the centres for these faculties. First, from the fact that focal lesions in the optic radiations of Gratiolet beyond the primary optic centres at the posterior termination of the optic tracts, abolish all three visual perceptions, he concludes that these primary optic centres do not bring optical impressions within the sphere of consciousness. Second, from the fact that in hemianopsia one of these faculties may be destroyed, the other two remaining intact, he concludes that these faculties reside in distinct centres, as no such condition ever results from disease of the chiasma or optic tract. Lastly, from the fact that colour-vision may be destroyed alone, or may be destroyed along with partial or complete destruction of form-perception, and that the limits of the destruction of light-perception are always either the same or less extensive than those of the two other faculties, from this he concludes that the fibres concerned in colour and form-perception must pass through the light centre. The visual centres he considers we have good ground for locating at the apex of the occipital lobe; but he does not decide whether the three centres for colour, form, and light are disposed in different layers of the cortex (the first being superficial), or side by side in the same layer. Dr. Wilbrand elaborates his theory in considerable detail, and illustrates it by a coloured plate. Each proposition he supports by the reference to the cases collated in Chapter II. Some of the details necessarily possess an *à priori* rather than an inductive character, and Dr. Wilbrand does not pretend that the proofs are in all points absolutely convincing. Even as a painstaking attempt to establish on a clinical basis a theory of the central relation of colour, form, and light-perception, to render definite what has been vague, the work is worthy of careful perusal. Its main value lies, however, it seems to us, in the second and third chapters, which may be read quite independently of the theory stated in Chapter I. The first of these two chapters is devoted to the classified record of cases of disturbance or destruction of colour, form, and light-perception, with an interesting account of amnesic colour-blindness. In Chapter III., Dr. Wilbrand discusses the differential diagnosis of lateral, *i.e.* homonymous hemianopsia as due to lesion of an optic tract, of the pulvinar, of the optic radiations, of the cortex, or of the two last combined. We subjoin a *résumé* of his conclusions. (1.) In lesion of one optic tract we have lateral, *i.e.* homonymous, hemianopsia of the opposite side, always absolute, *i.e.* involving all three visual perceptions, and nearly always complete, *i.e.* involving all the field of vision to the right or left of a vertical line through the fixation-point. Marchand's case of softening affecting the lateral part of one tract, shows that it is possible for the hemianopsia to

be incomplete. Subsequent amaurosis invading the other halves of the two fields of vision, indicates invasion of the chiasma, and is in favour of affection of the optic tract rather than central lesion. Pupillary reflex on illumination of the retinal area involved is absent, or there may be a tardy reflex due to dispersion of light. With the bilateral hemianopsia following invasion of the chiasma, the pupil is dilated and motionless to light. Photopsiæ may be present. (2.) In lesions of the pulvinar, the hemianopsia will be absolute and stationary, the pupil will react to illuminations of the implicated retinal area, and there will usually be temporary hemiplegia and hemianæsthesia. (3.) In lesions of the optic radiations, the hemianopsia will be absolute, but complete or incomplete; the pupillary reflex will be retained; the patient will often have photopsiæ, and symptoms of hæmorrhage, &c., will usually be present. (4.) In lesions of the cortex in the occipital region, the hemianopsia will be absolute or not absolute, complete or incomplete. A lesion of the tip of one occipital lobe may produce absolute and complete lateral, *i.e.* homonymous, hemianopsia. Destruction of colour-vision, with retention of form and light-perception, probably means damage just behind the external parieto-occipital sulcus, and a gradual extinction of form and light-perception would imply extension of the lesion towards the tip of the occipital lobe. In cortical hemianopsia, the pupil reacts promptly to illumination of the implicated retinal area; and when the lesion is double, and there is therefore bilateral hemianopsia, both pupils still react. The patient in such cases never has photopsiæ, and it is impossible to produce them.

JAMES ANDERSON, M.D.

De l'Aphasie et de ses différentes Formes. Par le DR. BERNARD.
1 vol. Paris: Delahaye, 1885. 8vo., pp. 270.

THE author bases his account upon the Lectures delivered in 1883 by Professor Charcot. He adopts and describes fully the four types of Aphasia known as Aphemia, Agraphia, verbal blindness, and verbal deafness, and devotes much attention to the history of the progress of science on this subject. His vindication of the claims of Broca is very vigorous, and reveals an important fact, viz. that the rights of Dax to the position now generally ascribed to him are by no means so firmly established as his Montpellier supporters have led us to believe. In fact, it is very doubtful whether the now famous memoir on the localisation of Language in the left hemisphere was ever read publicly, and it would appear that it first received publicity on the 25th of May, 1865. The whole discussion is worth reading (pp. 23 ff.). The history of the varieties of Aphasia called "sensorial," is not by any means complete. This is, no doubt, owing to the fact that the author, who appears to be master of the French language only, has been

unable to avail himself of the writings of foreign authors. We regret the fact, for the history of verbal deafness could have offered him ample scope for vindicating unrecognised claims. We allude chiefly to the strange neglect in which Dr. Bastian's remarkable paper, published sixteen years ago,¹ has been allowed to lapse.

Wernicke (1874) and Kussmaul (1877) are everywhere mentioned as having been the first to describe senserial aphasia. And yet we find the English author expressing himself on the subject in no uncertain or ambiguous terms. He divides cases of Aphasia into four categories: Aphasia proper, Aphemia, Agraphia, Amnesia.

"In Amnesia there is an inability to recall words, *i.e.* they properly cannot be revived in the auditory perceptive centres . . . in this condition we obviously have to do principally with defects of the cortical grey matter rather than with defects of afferent or efferent fibres, connecting this with lower centres" (p. 478). And he relates (p. 215) a case in which the patient suffered from verbal deafness.

It is to be remarked that, for Dr. Bastian, the process of thinking in words involves the activity of the auditory centre; in other words, that a train of such thought implies a series of revived sounds of words. He has defended this view with much ability against Professor Bain and others, who lay great stress upon the so-called "motor images." We do not find that Dr. Bernard, in the discussion of the important subject of the relationship between speech and thought, has grasped the importance of the point involved. His assumption that destruction of Broca's convolution produces "motor amnesia" is gratuitous, even on the supposition of the motor nature of the process of thinking in words, or "inner speech"; for he has not proved that the motor cortical zone is the repository of experiences derived from *conscious* muscular sensations; nor does he even seem aware that, failing this, he is assuming the highly improbable factor of a clear consciousness of efferent nerve-actions. Our criticism implies, of course, that the word "memory," applied by the author to motor and sensory processes, has the same sense in both cases, and does not, in the first case, designate merely the result of *unconscious* nerve-action in the building up of co-ordinative mechanisms.

Another deficiency in the work before us is the insufficient attention paid to the distinction between cortical and commissural Aphasias. This topic has recently been fully worked out by Lichtheim² who, like most other writers, is unaware of Dr. Bastian's published views, and quotes Wernicke as entitled to priority on this point. And yet the former, in the paper already quoted, insists upon the existence of intercentral commissures and their significance in pathology.

¹ "On Loss of Speech in Cerebral Disease;" *Brit. For. Med. Chir. Review*, vol. 43, 1869.

² 'BRAIN,' January 1885.

"It seems almost certain that impressions from the organs of sense to the perspective centres in the cerebral hemispheres travel along definite routes, although we may be more or less ignorant as to what those routes are, and also as to the extent and situations of the ground occupied in the cerebral hemispheres by the general perceptive centres . . . we may be sure that such centres exist somewhere, and are in connection with afferent fibres. . . . And because strong associations grow up between impressions [in different centres] we are entitled to infer that abundant communications exist between any single centre and all the others" (p. 477) . . . and in the succeeding pages Dr. Bastian goes fully into the symptoms arising from interruptions in those communications. The latter topic is still more fully treated in the 29th chapter of his work on 'Brain as an Organ of Mind,' written, we are told (p. 619) in 1878, before the author had seen Kussmaul's recently published monograph.

To Dr. Bastian, therefore, belongs the undoubted credit of having been the first not only to speak of cortical sensorial centres, but also to draw the vital distinction between motor and sensorial, and between central and commissural Aphasia. Dr. Broadbent, in a very remarkable contribution on "the Mechanism of Speech and Thought,"¹ starting from this position, adopts the hypothesis of distinct perceptive centres, and shows reason to localise the auditory centre "in the marginal convolutions of the hemisphere," adducing instructive examples of what we now call, after the German authors, word-deafness. To Wernicke belongs the credit of having still further defined the localisation of the auditory word-centre, and proved its existence in the first temporal convolution.

Notwithstanding the criticisms we have passed over Dr. Bernard's book, we are certain that its perusal will repay all those interested in the question of Aphasia. It is full of interesting matter. Among other details we may mention a case, transcribed in the original Latin of 1673, in which an instance of word-blindness is accurately described. The patient could write in several languages, but could not read a word, even of what he had written himself:

"Quod mirum est, si nomen ipsi aliquod vel dictio scribenda traderetur, prout illum et orthographice in quocumque ipsi antea noto idioma scribere sciebat, scriptum autem propriâ licet manu legere, vel characteres distinguere et dignoscere non poterat."

A. DE WATTEVILLE.

¹ 'Medico-chirurg. Transactions,' vol. 55, 1872.

Topographical Anatomy of the Brain. By J. C. DALTON, M.D., Professor Emeritus of Physiology in the College of Physicians and Surgeons, New York. 4to, 3 vols. pp. 175; 48 plates. Philadelphia: Lea Brothers and Co., 1885.

THIS magnificent work not only reflects the highest honour upon its author, but does full credit to those whose artistic and typographical skill has been put to contribution in its publication. It consists of a series of splendid heliotypes taken from photographs of the brain and sections of the brain under different aspects. Every plate is accompanied with an outline-figure containing the references to the explicative text. All illustrations are natural size. The author gives a full account of the methods which he adopted to overcome the considerable difficulties encountered in preparing the brain so as to yield faithful photographs of its natural appearances. The fresh brain supported by the calvaria is immersed, with the base upward, in a fluid of a little less than its own specific gravity. The author recommends the mixture of sodium chloride solution (sp. gr. 1026) with a small quantity of glycerine, as the most serviceable for this purpose. The ventricular cavities are then injected with a warm solution of gelatine by means of a fine canula introduced into the infundibulum, which should be preserved entire for that purpose. The injection should be continued until the ventricles are moderately filled and the gelatine begins to exude between the crura cerebri and the hippocampal convolution; after which the brain is placed in a refrigerator and the gelatine allowed to solidify. This secures the normal condition of the internal parts.

The next operation consists in imbedding the brain in a mass of gelatine, of about its own consistency, in a metallic framework so constructed as to allow of successive sections in the horizontal or vertical plane. The arrangements for cutting the sections were not quite the same for the different series, but we have no space for the details. In cutting, the knife and the section were flooded with equal parts of glycerine and water. The sections were allowed to remain for twelve hours in a refrigerator immersed in the saline solution before being photographed.

The difficulties of photographing were most successfully overcome. The sections had to be imbedded in gelatine, or immersed in glycerine and chloride of sodium solution. In the latter case, the pictures were taken from the image of the preparation in a properly disposed mirror.

In the first volume (10 plates) the external convolutions are represented. It includes a good view of the insula and operculum, obtained by a longitudinal section parallel with the median plane; and one of the parts exposed by another longitudinal section 5 millimetres deeper than the base of the insula. The relative position of the internal parts comes out in a very clear and instructive manner in Vols. II. and III., which give respectively

fifteen representations of horizontal, and twenty-three of vertical transverse, sections. Each section is made 5 millimetres beyond the preceding, so that the study of the successive plates allows the reader to obtain a very clear idea of the relative position of parts in the highly complex mass of the brain. The simultaneous use of the horizontal and vertical sections will still further assist him. Prof. Dalton has spared no trouble to render the descriptions lucid, and to bring into relief the clinically important features of the parts. The reader must remember that these volumes deal with topographical anatomy only, and that the problems of the course of the fibres and all histological disquisitions are outside its scope. Though thus limited, the work will be found to hold a unique and important position. It is a question whether any anatomist ever investigated the organ on the same plan and in so systematic a manner as Prof. Dalton has with so much success. At any rate, no series of plates at all comparable to these has hitherto been published before. The heliotypes represent things exactly as they appear in nature; the white and grey matter, the winding maze of the convolutions preserve their life-like appearances in the plates; and whilst the faithful outline-figures enable the student to identify at a glance the various structures, he realises to the full their mutual relations of parts under aspects never presented in the ordinary processes of dissection and anatomical teaching.

A. DE WATTEVILLE.

Injuries of the Spine and Spinal Cord without apparent mechanical Lesion, and Nervous Shock in their Surgical and Medico-legal Aspects.—By HERBERT W. PAGE, M.A., M.C. Cantab., Surgeon to St. Mary's Hospital. London: Churchill. Second Edition, 1 vol. 8vo., pp. 397.

WE are glad to find that a second edition of this volume has already been called for. It deals with a very important subject, both from a medical and an ethical point of view; and even where the author's opinions may appear to go somewhat beyond the facts he adduces, their expression has largely contributed to elicit further investigation and discussion. Mr. Page's searching criticism of the ideas conveyed by the expression "Concussion of the Spine," have done much to pave the way to a more scientific view of the nervous disturbances which so frequently follow railway accidents. Under this name a number of incongruous elements were heaped up together; and nothing was easier for medical men giving evidence in a court of law than to fall into hopeless confusion and contradiction with themselves and with others, when submitted to a searching cross-examination. Spinal and meningeal hæmorrhages, compressions, inflammations on the one hand, mere functional disturbances on the other, besides possibilities of ulterior chronic

inflammatory or sclerotic processes developing without any apparent immediate lesion, not to speak of a respectable contingent of "neurasthenic" or hysteroid manifestations, were all included within the elastic boundaries of a single morbid type. What appears to be the truth is that in most cases, besides more or less apparent external bruises, the victim of a railway accident suffers from what has been more or less aptly called "traumatic neurasthenia," partly due to actual cerebral shock, partly to the emotional disturbance at the time of the accident, and to the ulterior anxiety attending the usually protracted legal proceedings. It would be an interesting point with reference to the alleged curative influence of good damages, to determine whether such patients recover more quickly in countries where the claims are smaller, and the worry and complications attending a law-suit are of less portentous magnitude than in England.

It is obvious that the amount of genuine nerve disturbance, as contrasted with what the patient puts on when examined, must always be a difficult point to define. Some differences of opinion must necessarily arise in many cases. But a more important point still, is to determine how far Mr. Page has established that organic changes in the cord never, or only quite exceptionally, follow a mere "jar" of the organ. That such an occurrence is much more rare than is assumed by Erichsen, seems evident—especially in the case of individuals who at no previous period of their lives have offered any evidence of an inherited or acquired neuropathic predisposition. But is it irrational to suppose that a shock may play the same part as over-use, and by exhausting nerve elements determine the outbreak of an impending organic process? We have read of cases, and seen at least one, in which, post-mortem, the cord presented alterations of which the exciting cause appeared to have been a mere shock without immediate lesion. At any rate, such occurrences as the recent death of a schoolboy from blows received on the back shows that apparently superficial injuries may lead to serious results; and we must not forget that, though ordinary "traumatic neurasthenia" has usually a tendency to recovery, there are well-authenticated instances where the patient has remained a martyr to his functional troubles for a long time, if not for life, after the accident with which his misery began.

The second edition of Mr. Page's book is chiefly a reprint from the first. In the third he will perhaps find it possible to modify the controversial tone of some of the chapters, and enforce his views by a more systematic arrangement of his facts and arguments. His main position is a strong one; and he may now safely remove the cause of truth from the agonosphere of personal opinions into the calmer regions of critical judgment.

A. DE WATTEVILLE.

Abstracts of British and Foreign Journals.

"How the Blind Dream."—By MR. B. G. JOHNS. (*The National Review*, April 1885.)—In the dreams of most persons a mental vision is vividly produced during sleep, in which they perceive their friends moving about and conversing as in the ordinary real business of life. Now it is very obvious that such a dream cannot occur to a blind man; he cannot recall form and colour of which he knows nothing, and which an ordinary person has gained through impressions on his retina; his dream can extend no further than can be furnished by the faculties which he has retained. He may recall a person or a place, but the recollection can only be commensurate with what he has obtained by the senses of touch, hearing or smell. A blind boy dreamed of his brother who was dead. He knew him by his voice, and he also knew he was in the fields with him, for he felt himself treading upon the grass and smelling the fresh air. His idea of a field could not possibly reach much beyond this. Another man dreamed he was in his workshop; he knew this by sitting on a box and by the tools which were in it. A blind tramp said when he dreamed it was just the same as when he was awake, he dreamed of hearing and touching.

Mr. Johns mentions the case of a man who dreamed of a ghost. This suggests a question of very great interest. Do the blind believe in ghosts, and if so, in what manner do they come, and how are they recognised? A ghost is an apparition or ethereal being, generally resembling some person known in the flesh; it cannot, however, be felt, for it is transparent; a bullet may pass through it, and if sitting in a chair, it does not prevent another person occupying the seat at the same time. It is therefore generally admitted with Herbert Spencer that touch is the only reliable sense as a test of reality, is the one indeed into which the others may be reduced. When Macbeth could only see the dagger but could not feel it, he called it a dagger of the mind. How then can a blind man believe in a ghost known only by hearing and touch? It seems to us a contradiction, and yet Mr. Johns has a ghost story. A blind man dreamed he went to a house where he met a comrade who had been

sent to prison, and he thus described his dream. "I heard a voice at the door, and I said, 'Bless me, if that ain't John,' and I took him by the sleeve, it was his shirt-sleeve I felt, and I was half afraid of him, and surprised he was out weeks before his time. Then (in my dream) I dreamt that he tried to frighten me, and make believe he was a ghost by pushing me down sideways, &c., after that I waked and heard no more." This is a very curious account of the blind man's state of mind; he recognized his friend, but the latter behaved in so strange a way as to make the blind man believe he was a ghost. The pushing him down sideways, however, does not suggest a spiritual being to an ordinary mind. It would be a matter of great interest if Mr. Johns, or other persons coming in contact with the blind, would make further investigations into the subject of ghosts as conceived by the blind. The well-established ghost, clothed in white and quite impalpable, can scarcely have a place in the blind man's imagination.

SAMUEL WILKS, M.D.

Abstracts of Papers on Diseases of the Nervous System that have appeared in the Guy's Hospital Reports, 1874-1884.

1. Insular Sclerosis of the Brain and Spinal Cord. By W. MOXON, M.D. 1875.
2. On Meningeal Hemorrhage. By J. F. GOODHART, M.D. 1876.
3. On Cerebritis, Hysteria, and Bulbar Paralysis. By S. WILKS, M.D., F.R.S. 1877.
4. On Unilateral Atrophy and Spasm. By FREDERICK TAYLOR, M.D. 1878.
5. Report on Cases of Tetanus treated in Guy's Hospital. By FREDERICK TAYLOR, M.D. 1878.
6. A Case of Disease of the Brain with Descending Degeneration of the Spinal Cord. By FREDERICK TAYLOR, M.D. 1879.
7. History of the Physiology of the Nervous System, taken more especially from the Writers on Phrenology. By S. WILKS, M.D., F.R.S. 1879.
8. Note on a Case of Tetanus.—By R. E. CARRINGTON, M.D., and G. A. WRIGHT, M.B. 1879.
9. Reflex Action in Diagnosis. By P. HORROCKS, M.D. 1881.
10. A Case of Symmetrical Softening of the Corpora Striata, followed by Bilateral Descending Degeneration with Secondary Anterior Poliomyelitis. By W. HALE WHITE, M.D. 1882.

11. On Hemianæsthesia. By S. WILKS, M.D., F.R.S. 1883.
12. A Case of Extensive Cerebral Softening with Descending Sclerosis in the Lateral Column. By G. N. PITT, M.A., M.D. 1883-84.
13. Saturnine Lunacy. By J. F. GOODHART, M.D. 1883.
14. The Theory of a Heat-centre from a Clinical point of view. By W. HALE WHITE, M.D. 1883-84.

1. This paper was the first English account of this disease. The author points out that the disease can be diagnosed with certainty : its diagnostic characters are :—I. A peculiar trembling of the head and limbs during movements, ceasing when the parts are supported, so that whilst in bed the patient gives no sign of his malady, but does when he attempts to move, thus differing from the movements in paralysis agitans which are more even, do not stop when the part is at rest, and are not accompanied by any extreme idiocy of manner. The movements of insular sclerosis are most like those of mercury poisoning, but differ in that they affect the head and neck, and have the character of embarrassed helplessness. II. Paralytic weakness, especially of the lower extremities. III. Contractions and rigidity chiefly of the lower extremities ; they bear direct proportions to the paralysis. IV. Nystagmus ; this symptom may be late, it is earliest in the cerebral form of the disease. V. There is little or no disturbance in power over the excretions. VI. As a rule, the electro-sensibility as well as the electro-motility are normal. VII. The words appear to cost too much pains, and are produced one syllable at a time, each syllable accentuated. VIII. There may be great enfeeblement of mind. Although, as might be expected, considering the diffuseness of the lesion, the symptoms vary ; still, amongst this variety, there is a constant set as above enumerated. It is most common at the ages of 25-45 ; it is very chronic in its course, and death is usually accidental. The morbid anatomy is evidently that of chronic inflammation scattered in a broadcast style, especially in the white substance of the nervous centres producing patches of deep grey tint. The minute anatomy is shown very well in the spinal cord, where new sclerotic tissue is seen to advance inwards from a medullary ray, and all stages of the chronic inflammation may be seen. The paper concludes with a description of eight cases, in two of which there was an autopsy.

2. Meningeal hæmorrhage may be due either to variation in the blood pressure, or disease of the vessels. Out of 41 cases, 28 had either hypertrophied heart or granular kidney. Probably the

condition of periarteritis of the cerebral vessels described by MM. Bouchard and Charcot as causing miliary aneurisms, and so cerebral hæmorrhage, is the same thing as Drs. Gull and Sutton have described as hyaline fibroid degeneration of the arterial walls, and which is associated with chronic renal disease. The theory, therefore, which ascribes cerebral hæmorrhage to anomalies in the vascular tension is probably correct. Syphilitic disease of the arteries is one cause of meningeal hæmorrhage. Aneurism of the vessels at the base is a common cause; it generally exists in young people, and is a sequel to embolism. Mere excitement, or a sudden fit of passion, may, by inducing great pressure within the cerebral vessels, cause death. Three cases due to purpura are recorded. This may or may not be the result of blocking the vessels of leucocytes. The most constant symptom of the fatal cases is coma; of those that do not die, rather persistent stupidity remains. Meningeal hæmorrhage is difficult of diagnosis from alcoholism; the general appearance must be taken into account. Cases undoubtedly get well, but care must be exercised to avoid all excitement. The prognosis must depend upon the suddenness of the onset (one case in ten dies instantly), the completeness of the coma, the condition of the heart, the hardness of the pulse, and the state of the arteries. When the death is sudden, probably the medulla is implicated, either by pressure, or some disturbance of the circulation in it.

3. In this paper Dr. Wilks places on record some cases of cerebritis. In instances of cerebritis a large part of the cerebral hemisphere is involved in the inflammatory process, including the grey matter on the surface, the medullary matter within, and sometimes the central ganglia. As regards symptoms, there is no paralysis; the patient is simply devoid of mental power; there is generally no other symptom than this torpor, even if the meninges are involved. In unilateral meningo-cerebritis there may be some paralysis of the opposite side of the body. A case illustrative of this is given, but the patient did not know that she was paralysed. In this paper the doctrine that Dr. Wilks has often taught is emphasised, viz. that the formation of the animal body in two halves, and with two sets of nerves, necessitates the separation of the nerve-centres whenever an independent action of the limbs is required. The splitting of the spinal cord is a necessity of this independent action, and from this again of necessity follows a separate cerebral ganglion to rule over each. The mode in which

the two sides receiving different impressions can combine into one is by means of the commissure, and the explanation is no doubt of the same kind as that of single vision with two eyes.

The spinal cord may pass into the same condition that the brain does in sleep, and this is probably the explanation of some forms of hysteria with paralysis, and in hysteria with movement, there is over-action.

4. A case is recorded of a woman who, when a child 5 years old, was in the hospital for paralysis of the left arm and leg. She was admitted again when 27 years old. The left upper was much smaller than the right, the arm was close to the side, and there was flexion at the elbow, wrist and metacarpo-phalangeal joints; there were slow constant movements of the fingers. The left lower was also smaller than the right. She died of phthisis. There was found an old lesion in the grey matter of the corpus striatum, more or less atrophy of the right hemisphere, crus, pyramid, and olivary body, of the left hemisphere of the cerebellum and left side of the spinal cord. The right side of the base of the skull was smaller than the left. The history of the case was particularly clear; there could be no doubt but that the disease in the corpus striatum was primary; the atrophy of the other parts of the brain, as well as of the osseous and muscular structures, secondary. Some cases of congenital paralysis will bear a similar interpretation. The conclusion of Cotard, that partial atrophy of the brain is not a primary affection, but the result of traumatic, inflammatory, or other lesions, is justified. In the case here recorded there was no secondary degeneration, because the internal capsule was not involved. The case is interesting in confirming anatomy by the atrophy of the opposite half of the cerebellum. Eight additional cases are collected at the end of the paper. Out of the nine cases, seven commenced at or before the fifth year. Some of the cases were ushered in by convulsions, some by exanthemata; the paralysis is equally right and left, but always more in the upper than the lower. Sometimes the muscles are much wasted; sometimes in a state of painful tonic contraction, with slow involuntary movements; sometimes in a state of spastic contracture. The majority were subject from time to time to convulsive seizures, with or without loss of consciousness. There was very often some mental deficiency. The points of diagnosis from infantile paralysis are the hemiplegic distribution, the absence of marked atrophy in most cases, or the hypertrophied condition of the muscles that may be present in

some, the co-ordinated spastic contraction, the electrical reaction, the fits and the mental condition. The relation of this spastic hemiplegia, with slow movements, to the newly-described athetosis is discussed.

5. In this paper the cases during eleven years are recorded. Out of 51 cases available for statistics, 8 recovered; the traumatic cases were found to be more acute than the idiopathic; recoveries occurred more amongst cases of slight injury than amongst cases of severe; more recoveries took place amongst the cases in which the tetanic symptoms followed late upon the injury than amongst those in which they came on quickly; a more slow and chronic type of disease was more frequent amongst cases of mild injury than amongst cases of severe injury. In 12 out of 28 cases in which a post-mortem was made, there was lung mischief, in 13 nothing special was found. Of the 8 cases that recovered, 1 had Calabar bean; 1, Calabar bean and chloral; 1, Calabar bean, chloral, and morphia; 1, chloral; 1, chloral and iodide of potassium; 1, opium; 1, curara; 1, nitrite of amyl. In most of the Calabar-bean cases the drug was very rapidly pushed, either by subcutaneous injection or by the mouth; one patient took 125 grains of the extract in thirteen days, on one occasion he had seven successive doses of one grain each at intervals of fifteen minutes; 8 cases had Calabar bean alone; 2 had Calabar bean and chloral hydrate; 2 had Calabar bean, chloral hydrate, and morphia; 12 had chloral, and one of these had iodide of potassium added; only 1 case was treated simply with opium; 4 cases had curara; 3 cases had nitrite of amyl. The drugs used unsuccessfully were, quinine—bromide of potassium, bromide of ammonium, succus conii, *Cannabis indica*, nicotine, atropine, and aconite. Each case depends so much on its own merits, that, before any rigorous conclusions can be drawn, the article must be read in extenso.

6. A boy, aged 6, fell on his head six months before admission to the hospital; since then he has complained of pain, has seemed stupid, and has vomited; has lost power over his legs, and become blind. After admission, he got slowly worse; the face became paralysed; tonic contraction of the arms set in. At the end there was difficulty of swallowing; he gradually became emaciated and died. At the autopsy the following parts of brain had a gelatinous appearance; the posterior part of the corpus callosum and the posterior lobe on each side continuous with it; only the

posterior part of the corpus striatum was affected; the optic tracts ended in a mass of gelatinous material occupying the posterior part of the optic thalami and geniculate bodies. Sections of the pons, medulla, and cord showed disease occupying just those seats usually occupied by secondary degenerations, but rather darker and more defined than usual. The nature of the gelatinous material in the brain is discussed. Tumour, inflammation, and embolism appear to be excluded, and the degeneration shows no evidence of what preceded it. The secondary degeneration in the pons and cord is very interesting, because the posterior part of the internal capsule only was affected, and it therefore supports the views of Flechsig. Histologically, the condition in the cord was remarkable for the entire absence of granule cells, the absence of dense connective tissue, the great development of nuclei in the sheath of the vessels, and the substitution of a material, colloid in consistence and granular in structure, for the nervous and supporting elements of the tissue.

7. In this very interesting paper Dr. Wilks gives an account of some of the writings of the phrenologists, and of the transactions of the Phrenological Societies, and shows that they were not altogether worthy of the derision which their views excited. Dr. Wilks says, "I cannot but be struck with the great object which the writers presented to themselves, and the mode in which they proposed to prove the truth of their doctrine. Their object was the same as is now considered most rational. They discarded the notion that the brain was to be regarded as simply associated with the mind and there left, but they looked upon it as a compound and complex organ. They were the first who replaced the old method of the anatomists of slicing up the brain, by unfolding and dissecting it. They showed that it was made up of parts, each having its own function." Reference is made to a very able paper in the 'Phrenological Journal' for 1836, by Mr. Watson, entitled, "What is the use of the Double Brain?" and it is shown by quotations that the author had anticipated the views of Dr. Wilks on the duality of the brain, owing to which we have a double consciousness. Dr. Wilks takes this opportunity of giving clearly and fully his views on this subject. The phrenologists were remarkably accurate in the position in which they placed the seat of language; thus, Gall says, "The manifestation of verbal language depends upon a cerebral organ, and this cerebral organ lies on the posterior part of the superior orbital plate." By

numerous quotations it is shown, that the phrenologists of the first half of the present century were far in advance of the physiologists in their ideas of the locality of a part of the brain concerned with language. The phrenologists were also more correct in their views about sleep than many scientific writers of their time. In proof of this, it is mentioned that Dr. Caldwell records the case of a girl, in whom the brain, owing to loss of some of the bone of the skull, could be seen to protrude during activity, and to recede during sleep.

This paper by Dr. Wilks is so full of interesting discussions and references, that the reader must go to the original, as it is impossible to abstract it properly.

8. In this case the cervical enlargement of the cord was flattened on the right side; a cavity occupied the right cervical anterior cornu for its whole length: there was a smaller but similar cavity on the left side in the lumbar region; the cavities contained a rough shaggy material of a reddish-brown colour. The borders of the cavity showed marked degeneration of the nervous tissue. There was considerable hyperæmia, and several extravasations of blood were present.

9. In this paper a very good account is given of the best methods of obtaining reflexes, and also the diseases of the nervous system are classified according as to whether the reflexes are unaltered, absent, or exaggerated. The author performed some original experiments for the purpose of showing that the myotatic contractions were due to the loss of tone in the muscles, and some impairment of the reflex loop. A frog's muscle was removed from the body, but could not be made to contract by pulling on its tendon, although it responded to mechanical irritation directly applied to the fibres; but if the nervous system was uninterfered with, the muscle being left in the body, it was easy to get myotatic contraction by pulling on the tendon. It is concluded that the reason why the first experiment failed was because of the loss of tone, because the author thinks the time experiments that have been performed are conclusive proof that myotatic contractions are not reflex. The theories for the increased production of myotatic contractions in lateral sclerosis are discussed. Directions are given for obtaining the various clonuses and the front tap contraction.

An experiment is described which showed that the fifth nerve

has in it no fibres which cause dilatation of the pupil on strongly stimulating a sensory nerve, but that this result is brought about by the third nerve, probably through inhibiting the tonic influence, kept up reflexly by this nerve upon the circular fibres of the iris. The oscillations of the pupil are about twice as many for far as for near distances; this oscillation is absent in locomotor ataxy. Exhaustive or extreme pain causes dilatation of the pupils. Thus they are dilated after an epileptic fit, or after having a tooth extracted. These facts are useful in diagnosing malingering.

10. A boy, æt. $6\frac{1}{2}$, ran away from school, but was shortly afterwards found lying in the snow, and when picked up, could not stand, having right hemiplegia. After this attack he seemed stupid, never spoke or read anything, and has always suffered from loss of control over the bladder and rectum. This case is too long to condense satisfactorily. The patient had various nervous symptoms, such as paralysis of various parts of his body, loss of intellect, &c.; but the chief interest lay in the extraordinary hyperpyrexia, which at first was not constant, but towards death gradually became more so. It was very persistent; cold baths had to be very frequent to keep it under; the highest temperature reached was 107° . At the post-mortem examination, the brain and cord were the only parts affected. There the only abnormality was a small soft patch, a quarter of an inch in diameter, at the anterior part of each corpus striatum. There was well-marked degeneration on both sides down the crura into the cord, taking the usual course; the inflammation had at some places spread into the anterior cornua. The question is discussed whether the change in the lateral columns is primary; but the author concludes that the change in the anterior cornua is secondary. The hyperpyrexia is discussed in his paper on the "Heat-centre Theory," in the succeeding volume of the Reports.

11. Dr. Wilks points out, that whilst hemiplegia produced by disease of the corpus striatum is not a paralysis of half the body, but only of parts of it, hemianæsthesia produced by a lesion of the sensory fibres would in most cases be not only a complete loss of sensation of the entire half of the body, but of the special senses also; but hemianæsthesia accompanying hemiplegia is rare and exceptional; whenever loss of sensation exists with hemiplegia, the lesion will be found to affect the back part of the internal capsule. Complete hemianæsthesia, such as is above mentioned as theoretically

possible, is unknown to Dr. Wilks. All the complete cases of complete hemianæsthesia that he has seen are functional, and get well. Bearing in mind the dual action of the brain, it is possible that the hemianæsthesia is due to the cessation of sensory function of one half of the brain, and the cases which follow on a blow may be due to some concussion affecting half the brain only. Many facts and arguments are brought forward to prove these two points. Cases are given to show, that on rare occasions, male patients may present the same symptoms of this nature as female.

12. A girl, æt. 2, admitted for right hemiplegia, left facial paralysis, double optic neuritis, nystagmus, unequal pupils and vomiting. On admission, the case was thought to be one of tubercular meningitis; but as the child lingered on in an almost moribund condition, the diagnosis of cerebral tumour was made. The child died about seven months after admission. At the post-mortem examination, in addition to the extraordinary emaciation, so great, that the superficial cervical plexus could be observed under the skin, there was observed extensive softening of the middle part of the right cerebral hemisphere; no cause whatever could be found for it. There was well-marked descending sclerosis of the crossed pyramidal tract in the cervical region on the left side: most of the organs of the body were tuberculous; but there was no evidence of tubercular meningitis, and although the right middle cerebral artery was small, there was no evidence that the softening was due to this, for it did not correspond to the distribution of the artery.

13. The resemblance between the cerebral symptoms of lead-poisoning, of alcoholism, and of paralysis, is pointed out. Also the importance of not confusing the diagnosis, because the treatment is different, although it is extremely probable that plumbism will give rise to general paralysis. The similarity in the pathology of the three conditions is very marked, all being characterised by wasting cerebral changes; and in the case of lead and alcohol the analogy may be pushed still further, for both lead to persistent contraction and thickening of the vessels with arterial spasm. Therefore opium, digitalis, nitrite of amyl and nitro-glycerine will be of use.

14. The author points out that nervous non-inflammatory pyrexia is present in the following maladies:—1. Tumour of the cord, especially of the cervical region. 2. Tumours of the brain, especially of the pons. 3. Hæmorrhage of the brain, especially of the pons. 4. Embolism of certain cerebral arteries. 5. Ill-defined

degenerative changes in the brain. 6. Insular sclerosis. 7. Locomotor ataxy. 8. Obscure nervous cases that die without any change being found post-mortem. 9. Obscure nervous cases that get well, "Hysterical pyrexia." 10. Mental disease. 11. Injury to the spine. 12. Injury to the brain. Lowering of the temperature is present in—1. Tumours of the brain; 2. Cerebral hæmorrhage; 3. Degenerative changes in the brain; 4. Mental disease; 5. Injury to the spine. Cases are given illustrating all the above conditions. The author shows that all the cases may be explained, by supposing that there exists on the cerebral cortex, near the fissure of Rolando, a calorific centre which presides over the thermogenetic tissues of the opposite side of the body, restraining their activity. The fibres from the centre proceed down, by means of the corona radiata near the central ganglia, through the crura, pons medulla and cord, decussating in their course. In the cases of organic disease of the brain accompanied by non-inflammatory pyrexia, the lesion destroyed these fibres in some part of their course, thereby causing a rise of temperature, owing to the inhibitory action of the calorific centre being cut off. In the cases in which the temperature fell, it was due to the fact that the inhibitory fibres were stimulated. The physiological side of the question is fully discussed, and it is shown that the clinical facts agree with physiological experiments. Whether or not the calorific centre is a genuine calorific one, or whether it be a muscular vasomotor one, is not yet decided; probably the former is the true view. In the cases of hysterical hyperpyrexia the explanation most probably is that there exists functional disturbance of the calorific centre.

W. HALE WHITE, M.D.

I. On a Diagnostic test for Sciatica.—DE BEURMANN (*Archives de Physiologie*, April 1884, p. 375). Place the patient in a recumbent position, keep the leg extended, and gradually flex the thigh on the pelvis. Pain is caused, and he resists the movement.

Flex the thigh in the same way, but with the leg flexed. No pain is caused, and no existence is encountered.

The pain is caused, according to the author, by the tension put on the sciatic nerve, which, as he shows by experiment on the dead body, is greater in the first manipulation than in the second. He thinks the manipulation described furnishes a diagnostic sign of pain due to inflammation of the nerve as opposed to pain from disease of the joint or of neighbouring structures, and that it

furnishes a hint for treatment, viz. that we should fix the limb in the position in which the nerve is most relaxed. [The process known as "subcutaneous nerve-stretching," is based upon the facts adduced as new by this author. The thigh is flexed on the abdomen, and then the leg straightened out, thus placing the sciatic nerve on the stretch.]

II. On the Changes in the Nervous System after Amputation.—HAYEM and GILBERT (*Archives de Physiologie*, May 1884, p. 430). Amputation of right forearm just below the elbow in 1876.

Death from phthisis in 1883, at the age of 47.

Brain healthy and symmetrical.

There were neuromata on the ulnar median and radial nerves at their terminations in the stump. These were composed of a large number of small nerve fibres, formed of an axis-cylinder covered with a thin layer of myeline, and grouped in fasciculi. The course of these fibres was very intricate, but for the most part their direction was the same as that of the fibres of the main nerve-trunk, till they approached the periphery of the neuroma; then they turned back in a looped fashion. Their relation to the healthy fibres of the nerve trunk seemed to be this:—that at different levels the healthy fibres perished and were each replaced by a number of thin small nerve-fibres, the actual point of continuity between the two kinds of fibres was, however, never observed.

The neuromata therefore appear to be the expression of a regenerative process; viz. the growth of new nerve-fibres from the cut-end of the nerve.

Changes were found in the nerve-trunks as follows:—The radial was slightly thinner than the nerve of the opposite side; the median, about the same size; the ulnar, considerably larger. All three were grey, rounded, and firm.

In the radial and median there was atrophy of nerve-fibres with growth of interstitial connective tissue. In the ulnar, two varieties of nerve-fibres were found. (1) Large isolated fibres, with a fine axis-cylinder and thick coating of myeline, accompanied by a longitudinal band of connective tissue; (2) fasciculi of small fibres with a comparatively large axis-cylinder, and thin coating of myeline. The interstitial elements of the nerve-trunks (neurilemma, perineurium, epineurium, &c.) were increased.

In addition, therefore, to the degenerative process found in the

other nerves, there were in the ulnar separative changes, analogous to those in the neuromata, indicated by the presence of the small fibres.

The anterior and posterior roots of the brachial plexus were atrophied, many of the normal fibres had disappeared, and there were more small fibres than normal.

Spinal cord.—In the *white* substance, the posterior columns of the affected side were smaller than those of the sound side; but for the other columns just the reverse relation held. Of the *grey* substance there was atrophy at the level of the brachial plexus (particularly at the level of the 7th and 8th cervical and 1st dorsal nerves), affecting chiefly the anterior horns, and principally due to atrophy of the cells; the number of which was in some places only one-fourth or one-fifth the number of those on the sound side. Careful search with high powers showed the relics of cells—small, regular in outline, but still presenting traces of their normal processes; even those that remained visible to a low power had undergone some modification; they were rounded, and had but few processes.

III. On the Nervous System of an Idiot. Anomalies in the Convolutions. Arrested development of the Pyramidal Tract in the Cord.—HERVOUET (*Archives de Physiologie*, 1884, p. 165).

Age of patient $3\frac{1}{2}$ years.

Atrophy of the frontal lobes, with thickening and adhesion of the meninges, superficial softening with more deeply seated induration of the convolutions.

On the left side of the brain (which was principally affected), all three horizontal frontal convolutions were atrophied, the superior in the highest degree; the ascending convolutions were poorly developed, the paracentral lobule was small. On the right side the changes were less marked.

In the spinal cord were the following abnormalities:—

In either lateral column was an area coloured brightly by carmine, and characterised by absence or paucity of nerve-fibres; this area corresponded roughly to that of the crossed pyramidal tract, but differed from it in shape (it had not the usual triangular shape), and in its greater extension in a forward and backward direction. There was, besides, atrophy (or non-development) of the columns of Goll, and of the right direct pyramidal tract.

During life there had been no spastic symptoms. From this fact, from the unusual area of the tract of lateral disease, from

the simultaneous disease of the columns of Goll (without focal lesion to have originated ascending degeneration), the author concludes that the disease of the lateral columns was not secondary to the disease of the brain, but was due to arrested development of the cord.

This is borne out by his investigations into the development of the healthy cord; for he finds that even in healthy children an area of the lateral columns corresponding to that observed in the present case, does not reach perfection till the age of 4 years. But, though the question is only one of degree, yet in no healthy child of $3\frac{1}{2}$ is the development so backward as in this idiot.

IV. On Radial Paralysis. Theory of Nerve-compression. JOFFROY (*Archives de Physiologie*, May 1884, p. 478).

The author seeks to establish the truth of Panas' theory of paralysis of the radial from compression, in cases where, at first sight, its applicability might be questioned.

The case he gives is that of a man in whom paralysis of the left radial came on suddenly, after carrying a weight of about 50 kilograms, strapped on like a knapsack. The weather was warm, he had experienced no chill, his previous health had been good. But in order better to support his burden he had been proceeding with his forearms crossed and the right hand clutching the lower end of the left humerus. In this way he had, says the author, compressed the left radial at the point where it comes out between the triceps and the supinator longus. He explains in a similar way a case narrated by Duchenne, where a woman went to sleep in a chair with her arms folded in front of her.

The radial nerve itself is superficial, and can be compressed against the bone at the lower part of the humerus; but it gives off a branch to the triceps, which is partly covered by muscular fibres; consequently where this branch is compressed too, the triceps is also paralysed, though this is rare, and the paralysis is slight in degree, because this branch is partly protected from pressure by the covering of muscle.

On a Case of Transverse Myelitis. RAYMOND and ARTAUD (*Archives de Physiologie*, January 1884, p. 114).

Male patient, age 47. Very doubtful history of syphilis. In August 1882 left leg began to drag; some days later, sudden but transient failure of power in both legs while walking; then increased loss of power in left leg. Next, loss of power in con-

trolling evacuations, and incomplete paraplegia. The whole came on within a few weeks. Antisyphilitic treatment by MM. Charcot and Lasègue produced no benefit. Within six weeks he was admitted to the Hospital for Incurables, under the author. There was then complete paraplegia of the flaccid type, with anæsthesia to all forms of sensation, with abolition of tendon reflexes, and partial preservation of skin-reflexes; crossed reflex on tickling the feet. Farado-contractility in thigh-muscles feeble, in muscles of legs almost absent. Bedsores on heels, inner aspect of knees, and sacrum. Death from pyæmia six months later.

Post-mortem.—No lesion of vertebral column or meninges. *Spinal cord.*—In *mid-dorsal* region transverse myelitis, leaving unaffected only a few fibres round the anterior cornua and between the posterior cornua. All the constituent parts of the cord were (with these exceptions) involved indiscriminately at this level.

Above and below the mid-dorsal region the *grey matter* recovered its normal appearance; but in the white matter there was—*above* the lesion, degeneration of the columns of Goll reaching to the upper part of the cord; of Burdach's columns, ceasing rapidly as the sections passed up the cord; of the cerebellar tracts, to the upper part of the cord; *below* the lesion, degeneration of the crossed pyramidal tracts reaching to the lumbar region.

The adductor muscles of the thighs were in a state of degenerative atrophy. The spinal ganglia and sciatic nerves were normal.

The authors think that the lesion was not syphilitic, as there were no other syphilitic lesions, and anti-syphilitic treatment did no good. The myelitis is best classed as a sub-acute myelitis. The acute decubitus is to be connected with the extensive transverse lesion of the cord. There were no symptoms pertaining to the upper limbs.

VI. Hemiatrophy of the Tongue in Tabes Dorsalis. BALLET (*Archives de Neurologie*, vol. vii. (1884), p. 191).

Atrophy of muscle, irregularly distributed and not regularly progressive, occasionally complicates tabes. Hemiatrophy of the tongue has been noticed by Charcot (and also by Ross and Erb) as an early symptom of tabes.

In addition to a case by Cuffer (for which see next Abstract) the author mentions three cases.

1 (under Vidal). Male, age 17 at time of onset. The affection began in 1863 with sudden numbness of the left arm. Afterwards paralysis of the left side of face and left auditory nerve (sudden

onset), of the left 6th nerve; and difficulty of speech and swallowing. In 1866 pains in the limbs and arthropathy of the left shoulder. When the observations were made in 1875 there was—atrophy of the left half of the tongue (the taste and tactile sensibility remaining normal), paralysis of the left auditory nerve; wasting, with absence of farado-contractility in the left shoulder-muscles; arthropathy of the left elbow-joint, and to a less degree of the left shoulder.

2. Female patient at the Salpêtrière, age 51. In addition to the usual symptoms of tabes (the diagnosis of tabes was confirmed post-mortem), there was—arthropathy of both knees and of the right shoulder, paralysis of the internal and external recti of both eyes, atrophy of the left half of the tongue.

3. Male, age 35, still under observation. Besides the ordinary symptoms of tabes there was—atrophy of the muscles of the hands, and to a certain extent of the arms and of the sacro-lumbalis; paræsthesiæ in the district of the right fifth nerve; atrophy of the right half of the tongue. The affection of the tongue gave rise to very little trouble in mastication, swallowing, or speaking.

Hemiatrophy of the tongue is rare (the author says) in every form of disease except tabes (putting aside injuries to one hypoglossal), but pretty frequent in tabes; it should therefore lead us, when it is present, to suspect the presence of tabes. He ascribes the symptom to lesion of the hypoglossal nucleus, and notes the association with it (α) of ocular paralysis; (β) of atrophy of muscles in the limbs and trunk. He thinks that in these cases the sclerosis in the cord spreads from the posterior columns to the anterior cornua, via the commissural fibres described by Gerlach and Kolliker. In the medulla, the sclerosis spreads by a similar path (not yet anatomically demonstrated) from the root of the trigeminus to the motor nuclei of the medulla. From this point of view he emphasizes the existence (in his last case) of paræsthesiæ in the district of the right trigeminus; and he thinks that nervous disturbances in this area would be found in all cases of tabetic hemiatrophy of the tongue, if carefully looked for.

VII. Hemiatrophy of the Tongue in Tabes Dorsalis.

RAYMOND and ARTAUD (*Archives de Physiologie*, 1885, April 1, p. 367).

The case was originally described by Cuffer in 1875. Tabes had then existed for eight years; though for four years the symptoms had been limited to girdle-pains. There had been difficulty of

speech and of moving the tongue for a year. Slight atrophy of the right thenar eminence was made out, and well-marked atrophy of the right half of the tongue, with fibrillary contractions.

When seen again by Raymond in 1883, the most striking symptoms were as follows:—

General emaciation (though the farado-contractility of the muscles seems to have been preserved). Atrophy of the right half of the tongue. Deglutition normal. Speech comprehensible, though the articulation of some words was bad. No other bulbar symptoms, except immobility of the pupils.

Post-mortem.—There was sclerosis of the posterior columns, most extensive in the dorsal region, where it involved the posterior cornua and extended even to the anterior cornua, the cells of which were atrophied. In the cervical region the antero-internal group of motor cells, especially on the right side, was atrophied.

In the medulla, the principal nucleus of the hypoglossal, on the right side only, was atrophied. The accessory nucleus of the hypoglossal (= antero-lateral nucleus of Clarke, reticular formation of Stilling) was normal on both sides. The motor nuclei of the mixed nerves about the same level (vagus, glossopharyngeal, spinal accessory) were slightly affected, and less on the left side than on the right. All the other nuclei were normal.

In the right half of the tongue, the muscular fibres were much reduced in number, and fatty tissue had taken their place. In the trunk of the right hypoglossal many fibres were normal, but in others there was segmentation of the myeline, with disappearance of the axis-cylinders.

J. A. ORMEROD, M.D.

B R A I N .

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Original Articles.

ON A MUSCULAR PHENOMENON OBSERVED IN HYSTERIA, AND ANALOGOUS TO THE "PARA- DOXICAL CONTRACTION."

BY PROF. CHARCOT AND DR. RICHER.

PROFESSOR WESTPHAL was the first to describe, in 1878, an interesting muscular phenomenon, to which he gave the name of "Paradoxical muscular contraction." It may be described as follows:

A muscle is capable of passing into a state of tonic contraction by the fact alone of the bringing together of its points of attachment; in other words, of its relaxation. For instance, when the patient is lying on his back, the foot is suddenly flexed, and remains in that position by the contraction of the anterior tibial muscle, the tendon of which is seen to protrude under the skin. A gradual flexion may give rise to the same phenomenon. The foot remains in the position given to it during a more or less considerable time—27 minutes in the case of Westphal—then return to its normal position uniformly or by jerks. On repeating the process several times, the contraction ceases to occur: a result which may be attributed to muscular fatigue.

The phenomenon of the "paradoxical muscular contraction" may occur in consequence of the faradisation, direct or indirect, of the tibialis anticus muscle, or even in consequence of voluntary impulses. Professor Westphal considers it as the consequence of a purely passive factor—hence the term

paradoxical—viz. the slackening of the muscle; and opposes it to the ankle-clonus, which is produced by stretching of the gastrocnemius.

Dr. Erlenmeyer¹ has confirmed the clinical fact, but gives to it a different interpretation from Professor Westphal. He terms it a “contracture,” instead of a contraction; and, in the case of the tibialis anticus, he thinks that the excitation consists of the passive tension of the group of antagonists, viz. of the calf-muscles. As a proof of his statement he brings forward the fact, that the phenomenon cannot take place when, the leg being flexed upon the thigh, the calf muscles are pushed down with the hand towards their insertion into the heel, so as to prevent their being stretched.

Hence, there is no reason to speak of a “paradoxical muscular contraction.” We have to do with a simple reflex muscular contracture, resulting from an excitation of the antagonistic muscles.

Professor Westphal² answers that even on the supposition—a doubtful one—that a muscle can be shortened by the manipulation described by Dr. Erlenmeyer, the results of the experiment are to be attributed to the pressure upon the gastrocnemius, not to the approximation of its points of attachment.

Dr. Mendelssohn,³ whilst he confirms the clinical fact, explains the phenomenon by a rupture of the equilibrium of the tonus in certain muscular groups and their antagonists.

We have not had the opportunity of observing the paradoxical contraction in the various nervous diseases in which these authors say they have found it; but we have seen in certain cases of hysteria an analogous phenomenon, the analysis of which may throw light upon the question.

We have laid stress, in a recent communication to the “Société de Biologie,” upon a peculiar muscular condition frequently obtaining among hysterical subjects, which we have described as a “contractural diathesis.” It consists in a peculiar proneness of muscles to be thrown into a state of

¹ ‘Centralblatt für Nervenheilkunde,’ 1880, No. 17, p. 343.

² Ibid. No. 20, p. 317. See Abstracts, ‘BRAIN,’ Jan. 1881, p. 571.

³ ‘St. Petersburg. Med. Wochenschrift,’ 1881, No. 10

contracture under the influence of various stimuli, often of the lightest description. According to the mode of production of contractures, we have distinguished two chief forms of the diathesis: lethargic and somnambulic. These appellations are justified by the analogies with the two varieties of artificial contracture obtainable in hypnotism, according as the subject is in the lethargic, or in the cataleptic conditions. We must, for further details, refer the reader to the paper we have published on this topic. It will be enough, here, to state the fact, that in the more common of the two forms of contractural diathesis (the lethargic), Professor Westphal's phenomenon is most readily obtained. Here, by suddenly raising the toes, a contracture is immediately produced which keeps the foot in that position. A similar fact may be observed in the forearm, where a sudden bending of the wrist instantly determines a contracture, which fixes the articulation in flexion. The spasm subsides after a variable time; it may last several hours.

We wish to discuss here the mechanism of this contracture, which in our opinion has nothing "paradoxical" about itself. It is not, as Westphal believes, due to the sudden relaxation of a muscle, but is caused by the sudden tension of the group of antagonistic muscles. The following are our reasons for holding this view:

(a.) It is easy to convince oneself that in the contracture provoked by the sudden flexion of a joint (ankle or wrist), the attitude thus determined is not maintained in virtue of the action of a single muscular group, but is the resultant of two antagonistic muscular forces.

In the phenomenon observed on the foot, for instance, contracture affects not only the *tibialis anticus*, the action of which is dorsal flexion, but involves its antagonists, the *gastrocnemius* and *soleus*. It is easy to demonstrate this fact when the dorsal flexion is not complete. For as the impossibility of extending the foot is due to the contracture of the *tibialis anticus*, so the equally evident impossibility, of flexing it still further, depends upon that of the *gastrocnemius* and *soleus*.

(b.) It is well known that, in the patients we are speaking

of, contracture can be obtained by various manipulations. Malaxation, or massage, of the muscles is one of them. Thus if the tibialis anticus be kneaded, contracture soon sets in, and the foot is fixed in the state of dorsal flexion; massage of the gastrocnemius brings about the opposite result. In both cases the foot is drawn towards the muscle upon which the excitation has been made.

Under other experimental conditions, however, different results may be realised; and this is the point upon which we wish to draw the attention. If whilst the calf muscles are kneaded the foot is supported so as to be prevented from yielding to their contraction, we soon see it rise and assume a position of dorsal flexion, the more marked the more prolonged the excitation of the gastrocnemius and soleus.

A similar phenomenon is observed in the arm. If, whilst the extensors are kneaded, the hand is kept half flexed, the flexion soon becomes more accentuated by the contraction of the flexors, though the excitation be limited to the extensors.

Generally speaking, a stimulus applied to a muscular group acts upon the antagonists, the action of which acquires a predominance by the fact alone, that an obstacle is placed in the way of the movement of the limb in the direction of the excited muscles.

In other words, the antagonistic groups take a share in excitation applied to any particular muscle. The attitude resulting from the contracture does not depend only upon the seat of the excitation, but also upon external circumstances favouring or impeding the displacement of the limb in either direction.

The following explanation is consonant with the known facts of muscular synergy. The contracture being always a reflex phenomenon in the case we are considering, the afferent stimulus necessary to its production reaches the spinal motor cells innervating the excited muscles. This stimulus is thence conveyed to the cellular motor groups governing the antagonistic muscles through the commissural fibres which are universally admitted to exist. In order to account for the facts just described, we may assume that, whilst under ordinary

circumstances the antagonistic contraction thus obtained has only a regulating function, it may become preponderant when the effect of the direct contraction is, by some means or other arrested.

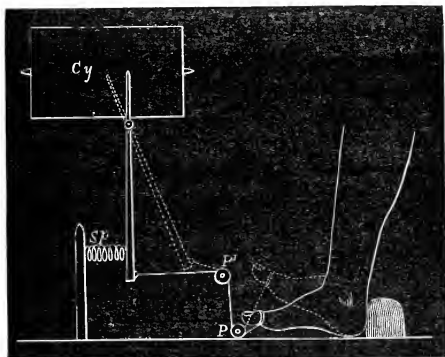


FIG. 1.—Method of experimentation :—To the foot, properly supported, is attached a string which passes over two pulleys, and is fixed to the end of a lever. This lever acts against a spring; and its opposite extremity, armed with a pen, records its movements upon the revolving cylinder of Marey's apparatus, going at the speed of one turn in thirty minutes. The dotted lines show the position of the foot, string and lever when the toes are raised by the contraction of the tibialis anticus.

Graphical curves obtained by the method of Professor Marey illustrated the following facts (cf. Figs. 1, 2, 3). With the exception of the beginning, which is more sudden when a contracture is produced by the flexion of an articulation, the

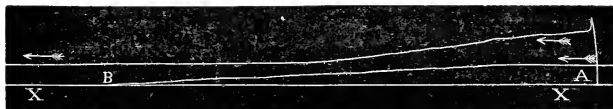


FIG. 2.—Tracing obtained (April 9th, 1883) in the case of C—, a hystero-epileptic, by the sudden dorsal flexion of the foot.—X X, horizontal line, or abscissa.—A, Beginning of the experiment.—B, total subsidence of the contraction.

curves are similar, whether sudden flexion or kneading be the exciting agency. The fall is sometimes interrupted by long

horizontal stretches. Now and then, especially towards the end of the experiment, staircase-like falls are noticed which correspond with more rapid local decontractions.

The duration of the contracture diminishes with the repetition of the experiment. Probably exhaustion would be thus produced in time. The later experiments show a more rapid line of descent, with more numerous falls.

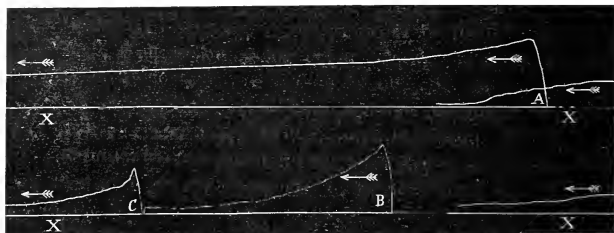


FIG. 3.—Same patient (April 12th). Tracing of contraction of the tibialis anticus obtained by massage of the muscles of the calf.—A, B, C, beginnings of three consecutive experiments. (In this and the last figure—much reduced in size—the length of X X represents one whole turn of the cylinder, viz. thirty minutes.)

When we compare these characteristics to those which we have found Professor Westphal attributes to the “paradoxical contraction,” a great analogy appears between the two sets of phenomena. The resemblance is further confirmed by Dr. Mendelssohn, who was present at our experiments, and found our tracings very similar to those he took himself from patients presenting Westphal’s symptom. There was the same suddenness of ascent, the same delayed descent with occasional falls. The only difference is in the duration of the phenomena, which in our patients occupy a much longer period.

The result we have reached is that the “paradoxical contraction,” as observed in cases of the contractural diathesis of hysteria, is due not to the sudden relaxation of a muscle, but to the stretching of its antagonist. Our opinion on this point coincides with that of Erlenmeyer and Mendelssohn, as will be seen by referring to the account we have already given of their views.

THE INERTIA OF THE EYE AND BRAIN.

BY JAMES M'KEEN CATTELL,

Assistant in the Psychological Laboratory, University of Leipzig.

INERTIA is a property of our sense organs. The molecules of the cells are only set in motion after they have been worked upon by a stimulus of a certain strength and for a certain time, and the motion continues after the stimulus ceases. In the case of sight, the lasting of the motion in the retina, and consequently of the sensation, after the stimulus ceases has long been known, and the phenomena of rotating discs and after-images have been carefully investigated. Less attention has been paid to inertia in the retina and in the brain when the sensation is excited. In considering this subject we must distinguish four operations, the time taken up by each of which we might seek to determine; (1) The time a light must work on the retina, in order that a sensation may be excited. (2) The time a light must work on the retina in order that the maximum intensity of the sensation may be brought about. (3) The time required for the light to be changed into a nervous impulse; and (4) The time taken up in the nerve and brain before the light is seen. Confusion has resulted because these operations and the times taken up by them have not been hitherto distinguished.

Plateau¹ incidentally remarked, fifty years ago, that a certain time is necessary "for the complete formation of an impression produced by a light;" but the subject was first taken up by Fick and Bruecke. Fick,² by an analysis of the law of Talbot and a few experiments, showed that a light must work on the retina for some time in order to call forth the maximum intensity of the sensation, and Bruecke³ found this time to be .186 s.

¹ 'Bulletin de l'Acad. de Bruxelles,' 1835.

² 'Archiv f. Anat. und Physiol.,' 1863.

³ 'Berichte d. Wiener Acad.,' 1864.

Exner,¹ with ingenious but complicated apparatus and methods, found the time to vary between $\cdot 119$ and $\cdot 287$ s., decreasing in arithmetical progression as the intensity of the light increased in geometrical progression. Kunkel² found this time to be different for different colours; with a moderate light, the colours being of about the same intensity, the time was, for red, $\cdot 057$; for blue, $\cdot 092$; and for green, $\cdot 133$ s. All these experiments tend to determine the time of the operation above numbered (2), but the experimentors did not always understand what time they were seeking to measure. Exner, for example, calls his paper 'Ueber die zu einer Gesichtswahrnehmung noetige Zeit,' confusing the operations (2) and (4). Experiments made by Baxt,³ under the direction of Helmholtz, should also be mentioned here. Baxt calls his paper 'Ueber die Zeit, welche noetig ist, damit ein Gesicht's Eindruck zum Bewusstsein kommt,' etc. (operation (4)), but does not at all determine this time. He allowed letters and curves to work upon the retina for a short time ($\cdot 005$ to $\cdot 017$ s.) and found that if soon afterwards a bright light was thrown on the retina, the letters or curve could not be distinguished. He found the interval which must elapse in order that the letters or curve could be distinguished depends on their nature and on the intensity of the second light, and thinks the length of this interval represents the time taken up in seeing the object, in which mistake he is followed by Tigerstedt and Bergqvist.⁴ The impression made by the object first presented may be erased on the retina by the strong light following; but if the times given are correct, it is probably erased in the brain centre, and the times determined represent about the difference in the apperception times of a letter or curve and a bright light.

I shall shortly print an account of experiments I have made, looking to determine the time taken up by the operations above numbered (3) and (4); in this paper I consider the operation (1): the time a light must work on the retina in order that a sensation may be excited. It has been found that about ten vibrations are necessary to excite the sensation of a

¹ 'Berichte d. Wiener Acad.,' 1868.

² Pflüger's 'Archiv,' ix.

³ Pflüger's 'Archiv,' iv.

⁴ 'Zeitsch. f. Biologie,' 1883.

tone, and perhaps twice that many to enable us to distinguish that tone.¹ In the case of sight, experiments are not necessary to prove that this time depends on the intensity of the light. We can distinguish an object illumined an extremely short time by the electric spark; on the other hand, the astronomer can see a faint star only after he has for a considerable time fixated the point at which he expects to find it. We may consequently assume that the greater the magnitude of the light vibrations, the less is the number necessary to excite the retina. It remains, however, a question of considerable scientific and practical interest to determine the time an object must be looked at, in order that it may be possible to see it, considering (1) the nature of the object, and (2) the intensity of the light.

I. APPARATUS AND METHODS.

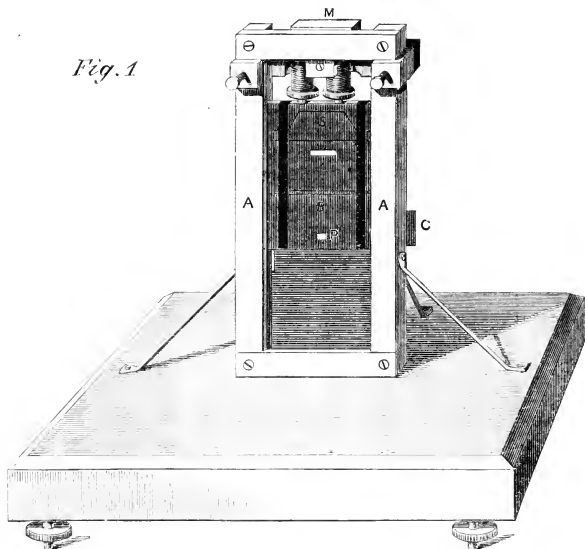
The apparatus I used, which may be called a gravity chronometer, is quite simple.² It consists (Fig. 1) of two heavy brass columns (A, A), 30 cm. high and 10 cm. apart. They are set exactly perpendicular by means of the three screws on which the base stands. Wedge-shaped grooves are worked into the columns, and in these a heavy soft-iron screen (s, s) slides without appreciable friction. The screen is 13 c. high, and has an opening 5 cm. wide, placed 5 cm. from the bottom. This screen is held up by an electro-magnet (M), which can be adjusted at any height desired. When the current flowing through the spiral of the electro-magnet is broken, the screen falls, falling always through the same distance in an exactly constant time. The object to be seen is fixed on a card (C), 15 × 3 cm., and this card is held in position on the columns by two springs, so that it is hid from the observer by the screen. A grey spot (P) on the black screen exactly covers the object to be seen, the spot being 3 mm. in front of the object. After a card, on which, for example, a letter has been printed, has been placed in the springs, the observer fixates the grey spot

¹ Wundt, 'Phys. Psy.' ii. 260.

² Carl Krille, Leipzig, has made the apparatus several times after my model, and charges about 4*H*. It may be examined at Leipzig, or at the Army Medical Museum, Washington, U. S. A.

on the screen, and by breaking the current which had been flowing through the spiral of the electro-magnet, lets the screen fall. The letter is seen at the point fixated, while the slit passes and is again covered by the screen. To determine the time the object has been in view, the screen is covered with smoked paper, and a tuning-fork is allowed to write on it as it falls. As the screen falls with the most perfect constancy, the determination with the tuning-fork need only be made so

Fig. 1



often that we are sure there has been no error. The theoretical time for a body falling in vacuo is scarcely shorter than the actual time for the screen as determined by the tuning-fork. In my experiments the slit in the screen was to be taken 1·3 mm., in order that the object should be in view ·001 s. As I could regulate the width of the slit in the screen to ·1 mm., the times were accurate to ·0001 s¹. I did not use a screen

¹ If greater accuracy than this is required, it would only be necessary to make

with a slit the width of which could be regulated ; but, for practical reasons, the screen above described, with an opening 5 cm. wide. The entire screen was covered (as shown in the figure) with black cardboard, in which was a slit of the desired width. There was nothing in the method of the experiments to annoy or distract the observer. He fixated the grey spot on the black surface, and at the instant most convenient to him allowed the screen to fall. The object was in view the time desired, at the point fixated, and illumined with a light to which the eye was adapted, and the observer found himself looking at a black surface, an impression having been made by the object on the retina. The object was illumined by daylight from a clear sky, or by lamplight ; the latter being the more convenient as it can be secured at pleasure, and its intensity can be accurately regulated. I used a carefully-made petroleum lamp with circular wick. The centre of the flame was 18 cm. from the object, the rays striking the surface at an angle of 55° . Of course no direct light from the lamp was allowed to fall in the eyes of the observer.

These experiments, though begun in America, have been carried out in the psychological laboratory of the University of Leipzig. Most of the determinations were made on B. (Dr. Oskar Berger), and C. (the writer). Care was taken not to strain the attention, or fatigue the retina. The retina was slightly more sensitive after a pause, but this and other possible sources of variation were avoided, and they are thoroughly eliminated by the large number of observations taken.

II. THE SENSITIVENESS OF THE RETINA FOR COLOURS.

I find that the time a coloured light must work upon the retina. in order that the colour may be seen, is different for the several colours. I used lights reflected from pigments. The colours were not, of course, fully saturated, but in these experi-

the columns taller, and let the screen fall from a greater height. On the other hand, levers can be applied decreasing, to any extent desired, the rate at which the screen falls; the same result can be reached by an application of the principle of Atwood's gravity apparatus.

ments it would have been difficult, and in many experiments it is impossible to use the sun's spectrum. It might further be suggested that it is quite as interesting to investigate the eye's relations to colours such as we find them in nature and art, as to the saturated colours of the spectrum. The colours taken were red, orange, yellow, green, blue and violet. They correspond most nearly to rouge 0, rouge orange 5, orange jaune 3, vert 0, bleu 1, bleu violet 4, in the 1^{er} cercle chromatique given by Chevreul in his 'Exposé d'un moyen de définir et de nommer les couleurs d'après une méthode précise et expérimentale.'¹ It seems most difficult to secure good red and violet in pigments: the red used was slightly dark, and the violet reflected some red light. The coloured surface was 3×1 cm. on a white background. As explained above, the card was placed on the springs of the gravity chronometer, the observer fixated a point immediately before the centre of the coloured surface and allowed the screen to fall. The coloured light worked on the retina for a period depending on the width of the slit in the screen. To determine how long the time must be taken, in order that a sensation may be excited, each of the several colours was taken separately, and, in addition, seven intensities of grey, including white and black. When the colour is looked at for an extremely short time it appears grey, yellow approaching the shade of the white card, violet appearing black, the other colours of various shades of grey. Series were made in which the colour to be investigated was placed about five times in the springs, shades of grey approaching in intensity the colour about five times; the observer of course did not know in any single case whether the colour or grey had been taken. After the light had worked on the retina the time desired, he decided whether he had seen the colour or not. When the time was taken sufficiently long he always named the colour, and when it was not there, said correctly "grey." On the other hand, when the time was taken very short, the observer saw no colour, and either always said "grey," or if he imagined he saw colour, was wrong as often as right. Thus it is easy to determine the time at which the colour is usually seen; a point at which it is seen about nine

¹ Paris, 1861; also 'Mém. de l'Acad.,' xxxiii.

out of ten trials. This point is quite constant, and can be determined to the ten-thousandth of a second. Table I. gives the results of the determination made on seven individuals with daylight from a clear sky, as also determinations made on B. and C. under various conditions to be explained below. In all the tables, as well as in the text, $\sigma = \cdot 001$ second is taken as the unit of time.¹

TABLE I.

	—	Red.	Orange.	Yellow.	Green.	Blue.	Violet.
B	Clear sky . .	1.1	0.7	0.6	1.1	0.75	1.75
C	1.6	1.1	1.25	1.6	1.5	2.5
H	1.0	0.6	0.75	1.25	1.0	2.25
L	1.25	0.75	1.0	1.25	1.5	2.5
P	0.75	0.6	0.6	1.25	0.75	2.0
S	1.75	1.1	1.25	1.5	1.5	2.75
T	1.5	1.25	1.25	2.0	1.5	2.5
		1.28	0.82	0.96	1.42	1.21	2.32
B	White following	10.0	6.0	6.0	7.5	7.5	12.5
C	7.5	5.0	6.0	7.5	7.5	12.5
B	Orange following	10.0	10.0	5.0	15.0	4.5	15.0
C	10.0	10.0	7.5	15.0	7.5	12.5
B	Blue following .	3.5	2.25	2.25	7.5	6.0	7.5
C	4.0	2.5	3.0	7.5	6.0	7.5
B	Lamp . . .	1.0	0.9	1.25	1.4	2.0	1.6
C	1.6	1.25	1.75	2.75	5.0	3.0
B	$\frac{1}{4}$ intensity	1.1	2.75	..
C	1.4	6.0	..
B	$\frac{1}{16}$ intensity	1.25	4.0	..
C	1.6	7.5	..
B	$\frac{1}{64}$ intensity	1.75	6.0	..
C	2.0	10.0	..
B	$\frac{1}{256}$ intensity	2.5	9.0	..
C	2.75	15.0	..

The table shows that light reflected from a coloured surface must work on the retina from $\cdot 6$ to 2.75σ in order that it may be possible to see colour. The time varies with the several colours and with the different observers; the order of the colours is, however, almost constant.² The retina is most

¹ In psychometric experiments the times are usually given in thousandths of a second, and it would be convenient to have a symbol representing this unit: σ is analogous to $\mu = \cdot 001$ m.

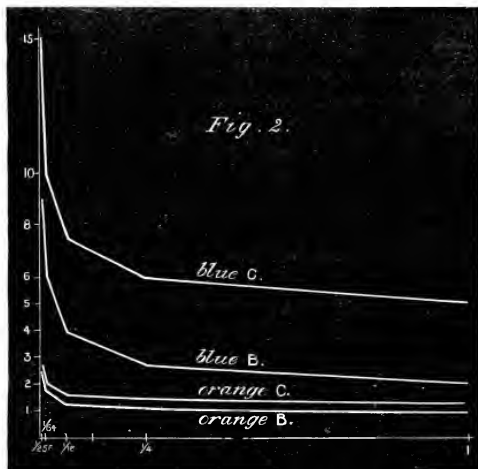
² It would be of interest to make these experiments on persons who are colour-blind.

sensitive to orange light, but nearly equally so to yellow. Blue must work on the retina about $\cdot 25 \sigma$ longer than yellow, in order that it may be recognised. Next come red and green, red requiring about $\cdot 1$ and green $\cdot 2 \sigma$ longer than blue. The retina is least sensitive to violet light, the time for which is two to three times as long as for orange. Undoubtedly the brightness of the colour has an influence on these times, but the brightness is a property not an accident of the colour, a saturated orange being brighter than a saturated violet. The time must be taken slightly longer if it is necessary to distinguish a colour from another lying near it in the spectrum, as orange from yellow, or blue from violet. I need not, however, give the results of the experiments I have made, as the times are but little longer than when the colour was to be distinguished from grey, and depend on the shade of the colours.

In these experiments, after the excitation by the colour the retina was allowed to rest, being only irritated by the amount of light reflected from dull black paper. If white paper (being illumined naturally by light from a clear sky) is substituted for the black, the light reflected from it washes away the impression on the retina, so the colour must work on the retina much longer (as shown in the table), in order that it may be distinguished. A coloured light following, lengthens in like manner the time, and the order in which the colours are seen is changed, as is shown in the table when orange or blue immediately follow the colour to be distinguished. B.'s retina is decidedly more sensitive than C.'s; his time is shorter for all the colours when the retina is not afterwards irritated; his retina is, however, also more sensitive to the irritating light, so that when this is present he must be given as long a time as C., in order that he may be able to recognise the colour.

When lamplight is substituted for daylight, the time for most of the colours becomes longer and the order is changed. The red light from the lamp makes orange considerably easier to distinguish than yellow, and places red next to orange. The time for violet, owing to the red rays it reflects, becomes shorter than for blue. I have given above (Sect. I.) the normal illumination with the lamp; if the lamp is placed twice as far from the coloured surface (the angle of incidence for the

rays being the same), the objective intensity of the light is reduced to one-fourth. I thus arranged five intensities of light, 1, $\frac{1}{4}$, $\frac{1}{16}$, $\frac{1}{64}$, $2\frac{1}{64}$, and determined the time for orange and blue (the easiest and most difficult colours to distinguish). The results are given in the table, and the curves (Fig. 2) are drawn, in which the abscisses are taken proportional to the intensity of the light, the ordinates to the time.



The curves approach both axes, but can reach neither of them, for on the one hand we can assume that even with an indefinitely strong light a certain number of vibrations would be necessary to excite the sensation, on the other hand we know that colour ceases to be visible before the light becomes indefinitely weak. The portion of the curve I have investigated follows the formula,

$$t = c \log i (+ c')$$

in which t is the necessary time and i the intensity of the light: that is, the time coloured light must work on the retina in order that it may be seen, increases in arithmetical progression, as the intensity of the light decreases in geometrical progression.

III. THE SENSITIVENESS OF THE RETINA FOR LETTERS AND WORDS.

Substantially the same method as for colours was used to determine the time the light reflected from a letter or word must work on the retina in order that the letter or word may be distinguished. After a card had been placed in the springs of the gravity chronometer, the observer fixated the grey spot on the black surface, and allowed the screen to fall. The light from the letter or word worked on the retina the time desired, and the observer either tried to name it, or said he did not know what it was. As in the case of colours, a point is found at which all the letters and words can be read correctly, another at which none of them can be seen, these two points being about half of a thousandth of a second apart. I tried to determine the interval at which half of the letters or words are correctly read. The time is quite constant, the normal variation being less than $\cdot 1 \sigma$. The sensitiveness of the retina is not, however, always the same; indeed, these experiments bear witness to a striking change. At the end of this investigation, the time for both B. and C. was about $\cdot 25 \sigma$ longer than at the beginning. This decrease in the sensitiveness of the retina was not gradual, but quite sudden. The change took place first in B.'s retina, so that for a time it was not more sensitive than C.'s. Then a corresponding and equally sudden decrease in the sensitiveness of C.'s retina took place. The following table contains the times for various classes of letters and words, the results being drawn from over fifteen thousand separate observations. The capital and small letters used were of the size in which this is printed. Twenty-six English and German words of four and five letters, and twenty-six of over eight letters were chosen. Each letter or word of the same class was placed five times in the springs, as a series (except in case of the German letters) was made up of one hundred and thirty observations. The table gives the times at which half of the letters and words are correctly read; the times being in some cases judged, from series in which more or fewer than half were read. The times given in the table

are for lamplight, they are about $\cdot 25 \sigma$ shorter for daylight from a clear sky. On the right-hand side of the table are given the results of experiments made on five sizes of letters taken from Snellen's 'Optotypi.' D is the distance in meters at which the letters fill an angle of $5'$, and is consequently proportional to the linear size of the letters.

TABLE II.

—	B	C	H	W	D =	B	C
Capital Latin letters .	1.1	1.4	1.6	1.2	4	.6	.7
Small " " .	1.1	1.4	1.7	1.3	1.75	.75	.9
Capital German letters .	1.25	1.7	1.7	..	1.25	1.1	1.35
Small " " .	1.15	1.5	1.7	..	.8	1.4	1.75
Short English words .	1.1	1.4	..	1.1	.5	3	4
Long " " .	1.2	1.5	..				
Short German words .	1	1.5	1.5				
Long " " .	1.1	1.55	1.6				

The table shows, that in order that a letter or word may be read, the light reflected from it must work on the retina from 1 to 1.7σ . There is a decided difference in the sensitiveness of the retinas of the four persons experimented upon, but the times for the several classes of letters and words do not vary greatly. The time is longer for the German (especially the capital) than for the Latin letters. This is due to the unnecessary complication of the German type and the similarity of certain letters. The time is slightly shorter for words than for letters. Children are now generally taught to read words as words, and are not required to spell out the letters; but it is well to prove that we read a word as a whole. The time is longer for long or rare words, and for words in a foreign language.

As in the case of colours, the impression left by the letter or word on the retina is washed away if it is followed by a white light, the time must consequently be taken longer in order that it may be possible to distinguish the letter or word. The times are given in Table III., when the capital Latin letters were followed by a white light lasting 1, 3, and 5σ and indefinitely. If the process started by the letter is allowed to work on for 5σ , the effect of the disturbing stimulus is not so

great, as the table shows. On the right-hand side of the table are given the results of experiments made to determine the relation of the intensity of the light to the time under consideration. Capital Latin letters were used, the method being substantially the same as that described above for colours. It will be noticed that the general form of the curves is the same as for colours, but the portion investigated does not follow the formula $t = c \log i (+ c')$, the ordinates t increasing more rapidly.

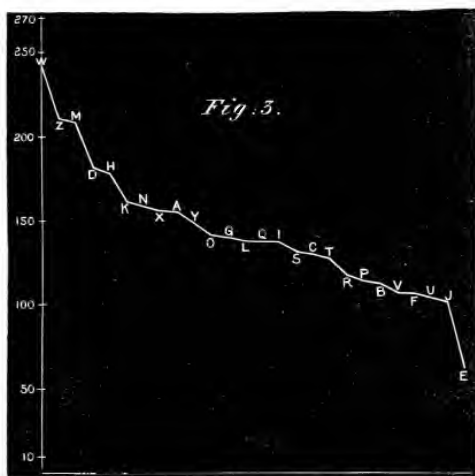
TABLE III.

—	B	C	Intensity =	B	C
White 1σ	1·6	1·7	1	1·1	1·4
„ 3σ	2·3	2·4	$\frac{1}{4}$	1·4	1·75
„ 5σ	4	4·3	$\frac{1}{16}$	1·85	2·5
„ indefinitely	6·5	6·5	$\frac{1}{64}$	4	6
Black 5σ , white 1σ	1·5	1·5	$\frac{1}{256}$	10	20
„ „ „ indefinitely	3·2	3			

IV. THE RELATIVE LEGIBILITY OF THE LETTERS OF THE ALPHABET.

In the foregoing section we considered the time various classes of letters and words must work on the retina in order that they may be seen, and found that some alphabets are harder to see than others. In this section it will be shown that the different letters of the same alphabet are not equally legible. These are both circumstances of the greatest practical importance. Reading is one of the largest factors in our modern life, but at the same time a thoroughly artificial act. Here, as everywhere in nature, the organism shows its power of accommodating itself to its environment, but the large percentage of children who become shortsighted and weak-eyed, and suffer from headaches, gives us sharp warning, and puts us on our guard, lest these diseases become hereditary. Considering the immense tension put of necessity upon eye and brain, it is of the most vital importance to relieve them by using the printed symbols which can be read with the least effort and strain. Experiments are not necessary to show that books (especially school-books) should be printed in large clear type,

but experiments, such as I have described, may lead us to determine the most favourable type. It seems probable that the use of two varieties of letters, capital and small, is more of a hurt than help to the eye and brain. All ornaments on the letters hinder, consequently the German type is injurious. The simplest geometrical forms seem the easiest to see. The lines must not be too thin; we seem to judge the letters from the thick lines, and it is doubtful whether it is advantageous to use thin and thick lines in printing. From all these considerations it seems that our printing-press has not improved



on the alphabet used by the Romans. Our punctuation marks are hard to see, and, I think, quite useless. It seems to me far better to replace (or at all events supplement) them by spaces between the words, corresponding in length to the pauses in the thought, or, what is the same thing, to the pauses which should be made in reading the passage aloud. Such a method of indicating to the eye the pauses in the sense would not only make reading easier, but would teach us to think more clearly.

As I have already stated, not only are some types harder to see than others, but the different letters in the same alphabet are not equally legible. It was found in making the experiments described in the foregoing section that certain letters were usually correctly read, whereas others were usually misread or not seen at all. Fifty-four series were made with the capital Latin letters, consequently each letter was used 270 times. Out of this number of trials, W was seen 241 times, E only 63 times. The relative legibility of the different letters is clearly shown in the curve (Fig. 3), in which the ordinates are taken proportional to the number of times each letter was read correctly out of the 270 trials.

I shall publish hereafter tables giving the number of times each letter was not seen at all, and the number of times it was misread, together with the letters for which it was taken. These tables show that certain letters, as S and C, are hard to recognise in themselves; others are mistaken for letters similar in form, as in the case of O, Q, G and C. The great disadvantage of having in our alphabet letters needlessly difficult to see will be evident to every one. If I should give the probable time wasted each day through a single letter as E being needlessly illegible, it would seem almost incredible; and if we could calculate the unnecessary strain put upon eye and brain, it would be still more appalling. Now that we know which letters are the most illegible, it is to be hoped that some attempt will be made to modify them. Our entire alphabet and orthography needs recasting: we have several altogether useless letters (C, Q and X), and there are numerous sounds for which no letters exist. In modifying the present letters, or introducing new forms, simplicity and distinctness must be sought after, and experiments such as these will be the best test.

Experiments made on the small letters show a similar difference in their legibility. Out of a hundred trials, d was read correctly 87 times, s only 28 times. The order of distinctness for the small letters is as follows: d k m q h b p w u l j t v z r o f n a x y e i g c s. As in the case of the capital letters, some letters are hard to see (especially s, g, c and x) owing to their form; others are misread, because there

are certain pairs and groups in which the letters are similar. A group of this sort is made up of the slim letters i j l f t, which are constantly mistaken the one for the other. It would not perhaps be impossible to put λ in the place of l, and the dot should certainly be left away from i (as in Greek). It seems absurd that in printing, ink and lead should be used to wear out the eye and brain. I have made similar determinations for the capital and small German letters, but these should be given up. Scientific works are now generally printed in the Latin type, and it is to be hoped that it will soon be adopted altogether. At present, however, it is impossible to get the books most read, Goethe's works, for example, in Latin type.

V. THE LIMITS OF CONSCIOUSNESS.

So far I have been speaking of inertia in the retina. I began by saying that the molecules of the cells are only set in motion after they have been worked upon by a stimulus of a certain strength and for a certain time; the experiments described, however, determine not merely the time necessary to set the molecules of the cells in motion, but the time the light reflected from a coloured surface or printed sign must work on the retina in order that it may be perceived. We found, for example, that a red light must work on the retina over $\cdot 001$ s. in order that the red may be seen. When, however, a red light works on the retina only $\cdot 001$ s., we have a right to suppose that the molecules in the cells are set in motion, and that a nervous impulse corresponding to the red light is sent to the centre for sight in the brain, but is too weak to excite there the sensation. We thus have to do with inertia in the brain, and the difficult subject of the threshold of sensation (minimum visibile).¹ It is not necessary to enter into a discussion of this subject in a paper, the object of which is to describe experiments, not to set up theories. I need only say that these experiments determine what may be called the threshold of consciousness.

¹ See Wundt, 'Physiologische Psychologie,' i. 321. Fechner, 'Psychophysik,' ii. 431. Exner, in Hermann's 'Physiologie,' iii. 2, 324. Funke, id. ii. 215.

There is not only a threshold of sensation, but also a maximum intensity of the sensation beyond which an increase in the strength of the stimulus does not increase the intensity of the sensation; analogous to this is a limit to the number of objects, or complexity of the object, consciousness can at one time attend to.

Whether the mind can attend to more than one thing at a time was a disputed question in the scholastic philosophy, under the form of the proposition: "*Possitne intellectus noster plura simul intelligere*;" and the subject has been discussed by a number of modern philosophers.¹ As the several philosophers reached their results chiefly through theoretical considerations, we need not be surprised that the number of ideas consciousness can hold at one time has been placed at from one to an indefinitely large number. Light is thrown on this subject by the number of feet of which a verse of poetry may be made up, and by the construction in music. Special experiments have been made by Wundt and his pupils,² from which it seems that when twelve to sixteen beats follow one another at intervals of $\cdot 2$ to $\cdot 3$ s. the number can be correctly judged, the separate beats not having been counted. In this case, however, it is probable that the beats are combined into groups, so that not sixteen simple, but fewer slightly complex impressions are at the same time present in consciousness. I have made experiments which show that the number cannot be correctly judged when more than four or five sound-impressions follow one another with great rapidity.

Experiments on the limits of consciousness can, however, be made to better advantage through the sense of sight. I have shown in a previous paper³ that from three to five (varying with the person) letters can be considered by consciousness at one time. This result is confirmed by an extended series of experiments I have made with aid of the gravity chronometer above described. In the first of these experiments, short perpendicular lines, 2 m. apart, were printed on a card, and, as above described with colours and letters, were allowed to be

¹ See references given by Wundt, '*Phys. Psy.*' ii. 214; and Hamilton, '*Metaphysics*,' i. ch. xiv.

² Wundt, '*Phys. Psy.*' ii. 213; id. '*Philos. Studien*,' ii. 3.

³ Wundt, '*Philos. Studien*,' ii. 4.

seen for $\cdot 01$ s.¹ Eleven cards were used, containing from four to fifteen lines, and the observer tried to give the number of lines on the card he had seen. The determination was carefully made on eight persons, and it was found that two could judge the number of lines correctly up to six, two up to five, three four, and one not four. This gives the number of simple impressions consciousness can at one time attend to. When the number of lines is larger than consciousness can grasp, it is estimated, those persons who can grasp the largest number estimate, as a rule, the most accurately. There seems to be a tendency to under-estimate the number of lines, and the mean error seems to follow the psycho-physical law, being directly proportional to the number of lines. Practice does not seem to improve the accuracy of the judgment.

In the same manner numbers, letters, words and sentences were exposed to view for $\cdot 01$ s., and it was determined how much consciousness can attend to at one time. The numbers and letters were printed with an American type-writing machine (Remington No. 4), five letters taking up 1 cm. The words were taken from a printed page, not more than two were put on a line, and they fell in all cases within the field of distinct vision. The sentences (likewise from a printed page) were divided into two lines when over three or four words in length, but extended in some cases beyond the field of distinct vision. The determinations made on eight individuals show a considerable personal difference, but on an average consciousness can at one time grasp four numbers, three to four letters, two words, or a sentence composed of four words. The letters are slightly more difficult to grasp than the numbers, every combination of numbers making a number that gives "sense." Not as many words as letters can be grasped at one time, but three times as many letters when they make words as when they have no connection. Twice as many words can be grasped when they make a sentence as when they have no connection. The sentence is taken up as a whole; if it is not grasped, scarcely any of the words are

¹ An object exposed to the retina for $\cdot 01$ s. can be seen very distinctly; if longer time is allowed, there is a danger that the impressions may be taken up by consciousness successively instead of simultaneously.

read; if it is grasped, the words appear very distinct; this is also the case when the observer constructs an imaginary sentence from the traces he has taken up.

In making these experiments I notice that the impressions crowd simultaneously into my consciousness, but, beyond a certain number, leave traces too faint for me to grasp. Though unable to give the impression, I can often tell, if asked, whether a certain one was present or not. This is especially marked in the case of long sentences; I have a curious feeling of having known the sentence and having forgotten it. The traces of impressions beyond the limits of consciousness seem very similar to those left by dreams.

The individual difference is a matter of special interest. B. out of forty trials read correctly five times a card containing seven numbers, and could always read five numbers correctly. He could grasp six letters, four disconnected words, or a sentence of seven words, whereas others could grasp but three letters, two words, or a sentence of four words. The latter numbers are the limits for one of the four students experimented on, and for the two women, one an educated young lady, the other the wife of a mechanic. The limit for a boy nine years old was somewhat higher. I tried to make the determinations on two rather obtuse porters, but their consciousness did not seem able to take up at all such delicate impression. They required three times as long as educated people to read a letter or word.

CASES OF OPHTHALMOPLÉGIA, COMPLICATED WITH VARIOUS OTHER AFFECTIONS OF THE NERVOUS SYSTEM.

BY JOHN S. BRISTOWE, M.D., LL.D., F.R.S.

THE cases, on which mainly this paper is based, were of long continued and great interest to those who watched their progress while they were under my care. They were cases of ophthalmoplegia; but, like so many cases of ocular paralysis, they were something more; and the other phenomena exhibited by them were probably even more interesting than those presented by the eyes.

The first case was in St. Thomas's for two years, and ultimately ended fatally there. But I did not know until, I think, after the death of the patient, that her case had already been published by Dr. Warner, in the 66th volume of the '*Medico-Chirurgical Transactions*;' nor did I know of some of the facts concerning her which he records, and which give additional interest to her case. His paper is entitled "*Ophthalmoplegia Externa, complicating a case of Graves's Disease,*" and the following is a brief abstract of it. Marion H. had had good health until February 1877, when the catamenia became scanty; and in November she was admitted under Sir Andrew Clark for tonsillitis, and was then found to present exophthalmos with considerable enlargement of the thyroid. In 1878 she was again admitted for Graves's disease, and suffered from palpitation, dyspnoea, bronchitis, and slight blood-spitting. The temperature sometimes rose as high as 103° without any inflammatory cause. Suffering from the above symptoms, she acted as a hospital nurse for some considerable time. About January 1880, she first suffered from diplopia, which lasted only for a few weeks. In November she began to notice that she was unable to move her eyes properly, and that to look at any object she had to turn her

head. She was admitted again in March 1881, and remained seven months under observation. During this time the signs of Graves's disease were present, but not excessive; she was very nervous and irritable; she had frequent attacks of palpitation, dyspnœa, headache and insomnia, during which the temperature often rose to 102° ; she also suffered from gastric crises, marked by vomiting, diarrhœa, epigastric tenderness, blood-spitting, and thirst. While in the hospital, she was much troubled with inflammation and ulceration of the corneæ; and presented double ptosis, with a double external squint, and incomplete paralysis of all the external ocular muscles. She seems to have improved in health under treatment, and the goître is said to have disappeared wholly. But the paralytic condition of her eyes and the proptosis remained without material change. It was thought that there was weakness of the 7th and 5th pairs of nerves, with general restriction of sensibility.

She was admitted into St. Thomas's about a couple of months after she left the London Hospital. At that time she had obvious but not extreme exophthalmos, double incomplete ptosis, and almost complete fixation of the eyeballs; but there was no defect of accommodation or of the action of the pupils, and the deeper parts of the eyes were healthy. It was soon discovered, also, that she had complete and absolute right hemianæsthesia, with colour-blindness of the right eye, and loss of smell and taste on the same side. But she had no loss of power in the right arm or leg, and could use them as well as the opposite limbs. It did not appear to us that there was involvement of the 5th or 7th pair. The thyroid was not obviously enlarged, and there was no evidence of disease in the thoracic or abdominal organs. She complained of headache in the occipital region, and on the left side of the head.

At this time I knew nothing of her having suffered from Graves's disease; and I was inclined to attribute her ophthalmoplegia externa and right hemianæsthesia to some degenerative change occupying the floor of the anterior part of the fourth ventricle and the walls of the iter, with extension into the neighbouring sensory tract on the left side, and her exophthalmos to paralytic weakness of the ocular muscles.

About two months after admission she had some inflammation connected with the right ear, attended with deafness; and she began shortly afterwards to discharge blood from this ear, and somewhat later from the right nostril. These discharges were continued henceforth to the end of her life; and six months before her death similar bleeding came on from the opposite ear. The exact sources of these hæmorrhages were never ascertained. But their persistence and abundance led me to suspect that my original view was wrong, and that there might be some slow-growing tumour in the situation where I had thought there was degeneration, and some similar growth implicating the dura-mater in the neighbourhood of the petrous bones, and invading the bones themselves. This opinion was not quite gratuitous; but was based upon the facts of a case admitted about the same time as this patient, in which the concurrence of discharge from one ear, with ocular and other paralyses, was found to be due to the association of a tumour springing from the floor of the fourth ventricle, with similar growths originating in the dura-mater of the several fossæ of the skull. The case is published in the 22nd number of 'BRAIN.'

In June 1882 the patient vomited for the first time while under my care; and from that time she continued to vomit for the most part two or three times a day. At the end of August in the same year she had an epileptic fit; and two months later a second, which was succeeded by loss of voluntary power and rigidity in the already anæsthetic right arm and leg. The paralysis and rigidity continued henceforth. In January 1883 she had her third fit; and, from that time, fits recurred every two or three weeks. It is important to observe that from time to time new nervous symptoms were added to those already present, but that no such symptom, of any importance, that had once developed ever subsided. During the patient's two years' residence in the hospital, she suffered as she had done, when under Dr. Warner's care, with ulceration of the corneæ; she was for the most part irritable and difficult to manage, and occasionally manifested delusions; and she had attacks of tonsillitis, and of bronchitis.

A very noticeable phenomenon in her case was the almost

constant presence of a temperature which ranged between 100° as its lower limit, and 103 , 104 or even 105° as its higher limit. This had no direct relation to her fits, and was not referrible to any inflammatory condition.

At the end of her two years, she seemed as well in general health as when she first entered the hospital; but she was suffering from headache, sickness, ophthalmoplegia externa, complete anæsthesia of the right side with rigid paralysis of the arm and leg, and repeated hæmorrhages from both ears.

What was the matter with her? I still concluded that the disease, whatever it was, occupied that portion of the brain which, in the first instance, I thought must be its seat. But I was divided in opinion between the presence of sclerosis and that of some kind of tumour. Against the existence of a tumour were, the absence of optic neuritis, and the fact that none of the cerebral nerves besides those of the external muscles of the eyes had become implicated. On the other hand, the persistent headache and sickness, and the involvement of the ears, seemed to me to point to tumour; and on the whole I leant to that view.

The patient went home; but just a month later was brought back to the hospital, moribund from an attack of bronchitis. I need scarcely say that the post-mortem examination was looked forward to with extreme interest. There were the evidences of the acute bronchitis of which she died. But the most diligent naked-eye search failed to detect even a trace of disease in the brain or cord, or any of the intracranial tissues. And, after hardening and staining, the most careful microscopic examination revealed no morbid changes whatever in any part of the cord, medulla, or mesocephale. There was no tumour, there was no recognizable degeneration. And, further, the hæmorrhage from the ears remained unaccounted for. But I am inclined to suspect that this part of the autopsy was not made with the same care as the rest of it.

It is a curious fact that a second case, clinically almost identical with the last, came under my care while this was still in the hospital. Early in January 1883, Gertrude H., a girl of fifteen, was admitted. She had been ill for about a month; and was suffering from headache, giddiness, paresis

of the external recti, and weakness and numbness of the right arm. The temperature was normal ; there was no optic neuritis, and the pupils acted to light and accommodation. By the autumn, without much change in other respects, she had lost voluntary power over all the external ocular muscles, and the eyes presented a downward and inward squint. Early in 1884, she was still suffering from headache and ophthalmoplegia externa ; she was giddy, and staggered in walking ; she complained of nausea, but had not been sick ; her right arm was weaker than it had been, and her leg also was weak ; further, there was impairment of sensation on the right side, mainly observed in the neck and chest, and in the area of distribution of the ulnar nerve. At the end of February, it was noticed that the tongue pointed to the right when protruded ; and, in March, she had an attack of left-sided chorea. While suffering from chorea she was sick for the first time, and a day or two afterwards had an epileptic fit. From this time onwards, she suffered severely from headache and giddiness, and from groups of epileptic attacks coming on every week or two, and preceded by aggravation of headache and sickness. Some time in May 1884, after one of her fits, her right arm and leg were found completely paralysed and rigid, the hand being clenched ; and they remained in this state henceforth. About this time, also, she had to take to her bed. When she was discharged from the hospital in February of 1885, her general health seemed fairly good. But still she had ophthalmoplegia externa, without any affection of the internal structures of the eyes ; her tongue was protruded to the right ; her arm and leg were not under her control, and more or less rigid ; her anæsthesia continued without much change ; and she suffered from headache and giddiness and periodical fits, the headache and giddiness for the most part coming on before a fit appeared.

The close likeness between these two cases is certainly very singular. In both there was almost complete ophthalmoplegia externa, in both there was paralysis with rigidity of the right arm and leg, and in both there was more or less complete right-sided hemianæsthesia ; moreover both patients suffered severely from headache and sickness, and frequent fits of an epileptiform

character. But there were also interesting, even if they were unimportant, differences between them. In the first case, there was constant bleeding from the ears; and there were also colour-blindness, and loss of taste and smell on the right side; all of which were wanting in the second case. And in the second case there was paralysis of the right side of the tongue, which was not observed in the other.

It is an interesting fact, too, that the progress of the second case was attended, as was that of the first, by frequent febrile rises of temperature. But, while in the first the elevation of temperature was more or less persistent, and had no apparent relation to anything in particular, in the other it was comparatively rarely present excepting as the forerunner of epileptic fits. The temperature in this case, as a rule, began to rise one, two, three, or even four days before a fit, and on the occurrence of the fit fell almost suddenly to the normal.

The nearly exact resemblance in respect of symptoms and progress of the second case to the first, makes it fairly certain that the resemblance extends to their ætiology and morbid anatomy, and that if the nervous centres of Gertrude could be examined, they would be found, like those of Marion, to all appearance healthy. It is of course impossible to say that there may not have been in the case of Marion minute structural defects in certain parts of the nervous system, which closer scrutiny, guided by a more profound acquaintance with pathology than we at present possess, might have enabled us to recognise. But the same may be said of cases of epilepsy, hysteria, and megrim, in which up to the present time no causative morbid nervous changes have ever been found. And on the same ground of morbid anatomy that justifies us in considering these to be functional diseases, we are justified, I think, in regarding as functional the affections for which Marion and Gertrude were under treatment. That there was something which it is customary to call neurotic in either case is shown by the circumstance, that both patients suffered at one time or another from functional nervous disorders, that had no apparent relation to the special groups of symptoms for which they sought my advice. Marion had for several years laboured under Graves's disease in a

well-marked form; and Gertrude was seized, while under my care, with an apparently imitative attack of chorea of short duration.

If the symptoms which my patients presented are to be looked upon as functional, may they also be regarded as hysterical? The answer to this question must depend, of course, on the meaning we attach to the word "hysterical." If every presumably functional nervous disorder occurring in women, to which as yet no other specific name has been given, is to be included in this term, then my cases were, perforce, hysterical. But the reasons for not regarding them as hysterical, in the common though somewhat vague meaning of the term, far outweigh, as it seems to me, the reasons adducible on the other side. It might, no doubt, be argued, that Marion's mental condition was exactly that which characterises many hysterical patients; that her hemianæsthesia resembled accurately the hemianæsthesia not unfrequently met with in hysteria; and that Gertrude's symptoms, following upon those of the other, and developing in the same ward, were imitated from them. But, on the other hand, Gertrude was a uniformly bright, sensible, placid girl, always grateful for whatever was done for her, and a general favourite with the nurses; hemianæsthesia, with involvement of the special senses on the same side, is not necessarily hysterical; and again, though Gertrude knew something of the other patient's symptoms, she did not know them all, or any of them accurately, and those in which she most resembled her were those she could not possibly have imitated, either consciously or unconsciously. The chief reasons, however, against the hysterical hypothesis are:—(1) the gradual and uniform progress of the symptoms from bad to worse (there was never any variability, never any shifting of paralysis or anæsthesia; whatever fresh symptoms accrued were permanent); (2) the character of the fits, which was clearly epileptic; (3) the remarkable prevalence of febrile temperatures without any obvious cause: and lastly, the character of the symptoms and their grouping, which formed a picture such as I have never read of as occurring, and have never seen, in any case of what has been termed hysteria.

Assuming the disease in either case to be functional, there is

still reason, I think, to believe that the functional disturbance on which the symptoms depended occupied mainly the region in which, during Marion's lifetime, I had assumed there was either progressive degeneration or progressive invasion by morbid growth—namely, the floor of the fourth ventricle and walls of the iter, with extension into the neighbouring sensory, and possibly even into the neighbouring motor, tracts. The hypothetical heat-centre lies in the neighbourhood; and, granting its existence, the explanation of the phenomenal temperatures present becomes easy. The relation of the rising temperature in Gertrude's case to the occurrence of fits reminds one of the similar sequence of phenomena met with in connection with the characteristic fits of general paralytics. In the latter instance, however, the rise of temperature is mostly, if not always, of comparatively short duration. May not the pre-epileptic rises, in the case of Gertrude, be essentially heat auræ?

A practical advantage in regarding the cases I have cited as functional is, that it fortifies us in the hope, so long as the survivor lives, that she may yet recover, and that many other cases of obscure and progressive brain-disease, which do not seem to be hysterical, and which simulate organic disease, may also prove amenable to treatment or the influence of time. I may here recall attention to a case in point, which I recorded in the last number of this periodical.

My third case is that of a man who was under my care for eight months, contemporaneously with Gertrude. His illness seems to have begun about five months previously. He first complained of drooping of the eyelids, and shortly afterwards of occipital headache, giddiness, and vomiting. He also suffered from what was called "inflammation of the stomach." While in St. Thomas's he laboured under occipital headache and giddiness; he had double ptosis and external squint; there were upward movement of the right eye, very slight outward movement of both eyes, and rotation of both eyes outwards and downwards, obviously effected by the obliqui superiores; the pupils were dilated and motionless, and he had no power of adjusting his eyes to distance; but in other respects his sight was perfect, and there was no inflammatory or degenerative

change at the back of the eyes; it was questionable whether there was any weakness of the 7th pair, or of the motor branches of the 5th, but there was marked impairment of sensation over the head and neck and upper part of the trunk. While under treatment, he suffered from occasional gastric crises, which were very severe, and once or twice of several days' duration; he had one or two epileptic attacks; and he suffered from frequent paroxysms of alarming dyspnoea, lasting from a few seconds to some minutes. The last were found to be due to paralysis of the abductors of the vocal cords. This patient seems to have had syphilis; but there was no evidence of secondary consequences.

Is this also an example of functional disorder of the nervous centres? If I had not had experience of the other two cases, I should unhesitatingly have attributed this patient's symptoms to sclerosis, affecting the nerve-nuclei in the floor of the fourth ventricle and iter, and extending downwards so as to involve the pneumogastrics and spinal accessories, and the sensory regions of the upper part of the cord. As it is, I confess I strongly incline to that explanation. At the same time, it cannot be denied that there is close resemblance between this case and the other two; and it is noteworthy that there were no symptoms referrible to the extremities suggestive of disease of the spinal cord.

I have added to my paper the brief details of two other cases of ophthalmoplegia, the one associated with wasting palsy, the other with locomotor ataxy; and in which, presumably, degenerative changes are in progress in the regions special to these affections, and in those presiding over the motor mechanism of the eyes.

CASE I.—*Graves's disease, followed by ophthalmoplegia externa, right hemianæsthesia, with involvement of organs of special sense, headache, sickness, and persistent high temperature, and subsequently by right hemiplegia, epileptic fits, bleeding from the ears, &c.—Death from Bronchitis.—Autopsy.*

MARION H., a single woman, formerly a hospital nurse, aged 25, was admitted into St. Thomas's under my care on the 18th of March, 1882.

She stated, that she had never had any serious illness until two years ago, when she had an attack of bronchitis ; that eighteen months ago she began to suffer from shortness of breath and palpitation ; and that four months ago she first observed drooping of the upper-eyelids, and double vision, which at the beginning were occasional only. She thought that her eyes had been unduly prominent for the previous twelve months. Four days before admission she took cold in her eyes from sleeping at an open window. There was no history of syphilis.¹

On admission, she was a well-nourished, and, on the whole, healthy-looking woman. She was complaining of headache, and of inflammation of both conjunctivæ. In addition to which there was marked prominence of the eyeballs, incomplete double ptosis, and almost absolute immobility of both eyes, which looked very nearly straight forwards. But the pupils were equal, and acted to light and accommodation. There was no enlargement of the thyroid body. She said she suffered from dyspnoea and palpitation at times ; but there were no present signs of these affections. The heart and lungs appeared to be healthy ; there were no indications of abdominal disease ; and the urine was normal. Tongue clean, appetite good, slept well.

For some weeks after admission she suffered mainly from ophthalmia, which proceeded to ulceration of the corneæ ; and for some days she suffered also from inflammation with excoriation of the tonsils. For the former affection she was placed under Mr. Nettleship's care, who found it necessary to stitch her eyelids together in order to ensure complete rest. During the time she was under treatment for the eye-affection no very minute investigation of her case was made in reference to other matters. It was noticed, however, that there was anæsthesia of the upper part of the right side of the face ; and it was hastily assumed that she had some affection of the fifth nerve, and the corneal ulceration was attributed to this circumstance.

In the early part of May, at which time the inflammatory affection of the eyes had in great measure subsided, the patient's condition was investigated with much greater care than had hitherto been possible ; and the following were the results, which were verified over and over again during the remainder of her life.

She complained of headache, which was variable ; sometimes being very severe, sometimes disappearing wholly, and referred either to the occipital region or to the region of the left parieto-occipital suture. There was moderate but marked exophthalmos.

¹ This history is inaccurate and incomplete ; but is corrected in the earlier part of this paper.

The upper eyelids drooped over the eyeballs so as to cover the pupils to a large extent; but could be raised (though very slightly) by the action of the occipito-frontales. The ptosis was incomplete; but the levatores palpebrarum could not raise the lids. The eyes looked very nearly straight forwards; could not be elevated or depressed; and could be moved outwards and inwards only within a very minute arc. The pupils were equal, and acted readily to light and accommodation. She saw double, but her sight in most other respects was good, and there was no sign of disease at the fundus of the eyes. There was absolute anæsthesia of the whole of the right side of the body up to the middle line. Nowhere on this side, neither in the conjunctiva, nostril, or mouth, nor in the face, nor in the arm, leg, or trunk, could she feel if she was touched or pricked, or if galvanism, heat or cold, was applied. The parts, however, looked healthy, and there was no difference in temperature, or as regards perspiration, between the two sides; and she had perfect voluntary power over the anæsthetic parts—she could move her arm and leg freely, could stand and walk without difficulty, and could feed herself with her right hand, and do needlework, so long as she saw what she was doing. Indeed, the readiness and accuracy with which she used her right arm and leg, made us doubt for some time whether or not the anæsthesia was real, or at any rate complete, the more so that she herself tried to conceal this infirmity, and consequently often answered questions about it untruthfully. There was a tendency for food to collect, unknown to her, in the right buccal pouch; and she stated on some occasions that, when drinking, the cup felt to her lips as if it were broken. The anæsthesia involved also the organs of special sense. She was never able to distinguish odours with the right nostril; nor could she at any time recognise the taste of sugar, mustard, salt, or any other sapid substance with the right half of the tongue. With her right eye she could distinguish forms quite as well as with her left, but was completely colour-blind; and while with the left she could sort coloured wools with the utmost nicety, with the right she failed to recognise any colours; and, without exception, when asked to put together those skeins which most resembled one another, selected the brightest scarlet and the brightest green. It was always very amusing to observe her endeavours, in the first place, to use the left eye surreptitiously when the right eye was being tested, and her look of disgust when on opening both eyes she found enclosed in her hand the inevitable red and green skeins. Indeed, she never would admit her colour-blindness, and always had some excuse to make for her error. There was no muscular paralysis excepting

of the ocular muscles; and the tendon-reflexes on both sides were normal.

About the middle of May, the patient complained of a painful swelling of the right cheek a little in front of the ear, and about the same time had a little watery discharge, tinged with blood, from the right auditory meatus. The swelling of the cheek soon subsided; but she suffered a good deal for the next few weeks from severe pain in the right ear and right side of the head; and soon had a pretty constant and pretty abundant discharge of blood, partly fluid, and partly clotted, from the ear; and she became deaf. I believe she was partially deaf of this ear previous to this attack. Mr. Clutton was consulted, and reported that the patient was suffering from acute external auditory catarrh; but he was not sure whether or not it was secondary to similar disease of the middle ear. The acute symptoms disappeared after a time; but henceforth to the end of her life she had an almost constant and abundant discharge of blood from the ear. Generally she passed a few drachms daily; but occasionally she went for two or three days or more without passing any, and under such circumstances usually complained of increasing headache, which was relieved when the discharge reappeared. Before long, blood came from the right nostril as well as from the right ear; and it was assumed that it reached the nostril from the Eustachian tube. It was never determined satisfactorily whether there was any perforation of the membrana tympani. Her deafness became aggravated after the commencement of the discharge; and soon the deafness on that side became absolute.

About the middle of June she began to vomit occasionally. The sickness recurred from time to time, but often at considerable intervals. On the whole, however, it increased upon her; and for many months before her death she vomited nearly every day, and sometimes several times a day. Nevertheless she maintained a good appetite.

Early in July it was noticed that she rambled occasionally; and towards the end of the month she complained, for many nights in succession, that an old woman, with something black over her head, was sitting by her bedside, and leaning over her. From this time onwards, and even to the end of her life, she was for the most part perfectly sensible; but she occasionally suffered from delusions; and became more and more irritable and exacting, not unfrequently flying into a violent passion, and using the grossest language towards the nurses and others who were waiting upon her.

On the 31st of August she had for the first time an epileptic

fit. She was generally convulsed; and passed water into the bed; but she did not utter a cry or bite her tongue. It lasted a few minutes. When she emerged from the fit her right arm and leg were found to be partially paralysed, the fingers being flexed; but there was no involvement of the facial muscles or of the tongue.

On the 25th of October she had a second fit of the same character as the first, excepting that it was preceded by a cry. It lasted about five minutes, and the patient went off into a profound sleep of several hours' duration. Subsequently she became delirious, violent and noisy, continually crying out, "My head, my head!" After this fit, the paralysis of arm and leg was complete, the two limbs were rigid, and the arm was kept extended while the fingers were strongly flexed. When the arm was raised from her side, it presented very rapid and very fine tremors. The condition of the limbs remained henceforth wholly without change. She never regained even a trace of power over them, and they were always rigid and finely tremulous.

The third fit occurred in January 1883, from which date the fits attacked her, not quite regularly, every two or three weeks. Sometimes they were solitary, sometimes in groups of two or three. They were often ushered in with a cry, and often her urine escaped from her during the attack. She once or twice bit her tongue, but was always more or less violently convulsed, for the most part equally on both sides, and very often at the moment when the fit was coming on threw herself out of bed on to the floor. The occurrence of fits was often preceded by increase of headache, and cessation for a day or two of hæmorrhage from the ear. Generally also about the time of the fits, and more after than before, the patient became noisy and fractious: and occasionally about this time suffered from hallucinations.

Very little change of any real importance occurred in the condition of the patient subsequently to her second epileptic fit, after which the right arm and leg became rigid as well as paralysed. All the symptoms of interest that were present on her admission, or had developed later, continued. But during her long residence in the hospital various minor complications arose. Her general health varied, and observations were from time to time made in confirmation or correction of previous examinations.

Among the complications referred to may be mentioned, first, an attack of tonsillitis with bronchial complication in November 1882; second, the appearance, about the end of March 1883, of bed-sores on the right buttock and sacrum, which, however, never attained a large size, and were healed in the course of a couple of months.

As to her general health, it may be mentioned that, towards the

latter part of 1882 and in the early part of 1883, she seemed to be losing flesh and strength; but that, subsequently, she improved in both of these respects, and then (excepting that she was pale from continued loss of blood) remained almost without change to the end of her life.

The state of the eyes was examined over and over again. The ptosis and exophthalmos presented slight changes from time to time, and were not always symmetrical; but there was never any definite improvement. The balls of the eyes were almost completely immovable, and looked very nearly straight forwards. It was generally noticed, however, that there were variable, and very slight, lateral movements in both; and that especially there was a slight degree of power in the left external rectus, and consequently an occasional slight outward squint of the left eye. There was sometimes observed a little inequality in the pupils; but it was confirmed that the intra-ocular muscles acted to light and accommodation. She could see distinctly with both eyes; but the fields of vision (and especially that of the right eye) were contracted. The colour-blindness continued in the right eye; but the left was never similarly affected. The fundus of the eyes remained healthy. The corneal ulceration and conjunctival inflammation were not finally cured until the end of August 1882.

The discharge of blood from the right ear and right nostril continued without abatement, even after all signs of inflammation in the outer ear had abated. Mr. Clutton believed that there was perforation of the membrana tympani. She became stone-deaf with this ear. In the beginning of October 1883, bleeding for the first time took place from the left ear also. And from this time forwards the discharge of blood from this ear, like that from the right, was nearly constant, though less copious. The hearing on this side also became impaired.

She never recovered feeling or smell in the right nostril, or feeling or taste in the right half of the mouth, including the lips, cheek, and tongue.

The anaesthesia on the right side of the body persisted. It is stated, however, in the notes that on some occasions there was slight evidence of sensation in the right foot.

The right-sided paralysis involved only the arm and leg, and never extended to the facial muscles or to the tongue. The paralysed limbs did not waste relatively to the others; their tendon reflexes, however, were somewhat more marked, and they occasionally presented both ankle- and knee-clonus. It was observed by Dr. Hadden, during the patient's stay, that the "paradoxical contraction" could be obtained in the paralysed limbs. Also,

the electrical reactions were investigated by Dr. Kilner with the following results:—

With faradism, all muscles require a strong current. With continuous current muscles of left side require a stronger current than those of right:—

Right upper arm .	7·500—	7·500+	Left upper arm .	3·500—	3·500+
„ forearm flexors	2·503—	·975+	„ forearm flexors	5·100—	5·100+
„ „ extensors	2·550—	2·000+	„ „ extensors	5·100—	5·100+
„ thigh . . .	5·200—	5·200+	„ thigh . . .	6·100—	6·100+
„ leg . . .	3·100—	3·100+	„ leg . . .	5·300—	5·300+

The headache, usually referrible to the occipital region, sometimes to the right side, sometimes to the left, and liable to severe exacerbations, continued during her whole illness; and for the greater part of her residence in the hospital she was sick once or more every day. Yet, notwithstanding this, she did not lose flesh.

A remarkable feature in her case was the almost constant prevalence of high temperature. Occasionally, and even for a few days together, it would go down to the normal. But almost always it ranged between 100 as the lower limit, and 103, 104 or even 105 as the higher limit. The cause of this was not apparent. It had no relation to the epileptiform attacks. There was never anything specially noticeable as regards the condition of the thoracic and abdominal viscera, the pulse or urine.

She was discharged on the 23rd of February, 1884, having been in the hospital just one year and eleven months; at which time she seemed on the whole as well, and as likely to live, as she had done a year previously.

On the following 25th of March she was brought to the hospital suffering from bronchitis, and in a moribund condition. She died early the next morning. It was ascertained that she had had several fits while at home, of which the last occurred a week before admission. The affection from which she died came on at that time.

Autopsy.—There was accumulation of mucus in the bronchial tubes, and distension of the lungs with air. A few granulations were found on the auricular aspect of the mitral valve; but this was neither contracted nor incompetent. The right side of the heart was somewhat dilated and thickened. The tonsils were large, with patches of secretion adherent to the surface. The uterus was retroflexed. All other organs in the chest and abdomen were healthy.

Calvaria, dura-mater and other membranes of brain healthy. There was no flattening of the convolutions; no affection of arteries or nerves; no congestion; no accumulation of serum, either in the

ventricles or in the subarachnoid tissue. And generally the substance of the cerebrum and cerebellum was healthy. On making sections of the floor of the fourth ventricle, two symmetrical dots of black pigment were seen, situated just below the surface, and immediately behind the corpora quadrigemina. And just outside that, on the left side, and a little deeper placed than it, was a larger patch of the same kind. The optic tracts, corpora quadrigemina, crura cerebri, and other parts in the neighbourhood all seemed healthy. There were, however, some doubtful sclerotic changes in the left anterior pyramid, and in the crossed pyramidal tract on the right side. The cord, medulla oblongata, mesocephale and other parts, were removed and hardened, and subsequently stained and sliced. A most minute and careful microscopical examination was made by Dr. Hadden and others; but nothing whatever was found indicative of morbid change. Every part appeared to be absolutely healthy; and the suspicions expressed with respect to one or two points at the time of the autopsy were not confirmed.

There was a great deal of fat in the orbits; and the ocular muscles were unusually pale, and seemed stretched. The right membrana tympani was perforated, but no disease of the middle or external ear was found on either side. There was some blood in the right meatus. But the source of the hæmorrhage during life was not discovered. The lobes of the thyroid body were somewhat large.

CASE II.—Ophthalmoplegia externa, right hemiplegia, headache and sickness, followed by partial right hemianæsthesia, and epileptic fits preceded by prolonged rises of temperature.—Chorea during the progress of patient's illness.—No result.

GERTRUDE H., aged 15, was admitted under my care on the 4th of January, 1883.

On the whole she had been a healthy girl; but had had fits from the age of 18 months to that of 5 years; and, about three months ago, a slight sore throat, which did not require medical treatment. Never had rheumatism or scarlet fever.

After suffering for about a week from giddiness, and headache referred to the right side, she was attacked suddenly on the 1st of December last with an internal squint of the right eye. And on, or about, the 30th of the month she first complained of weakness, numbness and tingling of the right arm. She had no sickness.

She was a pale, but healthy-looking girl; complaining of headache on the right side, double vision, and weakness and numbness of the right arm. She kept her right eye closed voluntarily;

because by so doing she prevented giddiness and saw better. There was obvious weakness of both external recti; but the left was distinctly feebler than the right; and she saw double when both eyes were open. The pupils were equal, and acted naturally. There was no affection of the optic discs. The right arm was partially paralysed, and the grasp of the hand was very feeble compared with that of the left. There was also some numbness in it; and it was thought that (though the patient did not acknowledge loss of feeling on the right side generally) there was less accurate tactile discrimination on this side than on the other. There was no facial or lingual paralysis, or paralysis of the leg; and no deafness, colour-blindness, or loss of smell or taste.

She sojourned in the hospital for two months; during the whole of which time her condition remained practically unchanged. The paralysis of the external recti and right arm persisted; she complained more or less constantly of pain on the right side of the head, and frequently of giddiness. She often suffered from nausea; but was sick on only one or two occasions. No affection of the pupils, and no optic neuritis, ever appeared. It was ascertained that the reason why she kept the right eye closed in preference to the left (which was the less paralysed one) was that the vision of the right eye, owing to short-sightedness and astigmatism, was less perfect than that of the left. There was no discovered disease of the abdominal or thoracic viscera. Her mental condition was good; but she was a little inclined to be low-spirited.

Her treatment consisted first in the use of tonics, later in that of iodide of potassium; and in the application on one or two occasions of leeches and counter-irritants to the temples.

On March 3rd, she was sent to a convalescent home, where she remained for a month without benefit. Shortly after her return, she came up to see me, when I found the right pupil dilated and immovable. This affection, however, was only temporary; and at the next visit the pupils were again equal and active. There was still no optic neuritis.

From this time to February 1884, she came to me as an out-patient at irregular intervals. Her general health remained much as it had been, and she continued to suffer from headache, referrible sometimes to the right side, sometimes to the back of the head, and occasionally extending to the back of the neck, variable in intensity and often very severe; from giddiness; from occasional nausea, but never sickness; and from weakness in the right arm. But the paralysis of the ocular muscles slowly extended; so that by the autumn there was paralysis of all those which move the eyeballs; and the eyes were fixed in the downward and inward direc-

tion, and the lids drooped. There was no affection of the muscles of the irides or of accommodation, and none of the fundus of the eyes. She still, as a rule, kept her right eye closed.

On February 5th, 1884, she was readmitted, and she remained in the hospital until February 2nd, 1885. The following is a statement of her condition at the earlier of these dates. She was a well-nourished, well-behaved, and happy-dispositioned girl. She complained of pain at the back of the head, and of giddiness, in consequence of which she staggered in walking. She suffered from occasional nausea, but not actual sickness. The right arm was weak, and the grasp of the hand much less powerful than that of the other. The right leg also was somewhat weak. There was some impairment of sensation on the right side of neck and upper part of right side of chest, and in the distribution of the right ulnar nerve; and generally also over the right side the power of localising impressions was imperfect. No wasting of muscles, no rigidity. The knee-jerks were exaggerated; and ankle-clonus was obtainable on both sides, but chiefly on right. There was slight double ptosis, and almost complete immobility of the eyeballs, which looked downwards and inwards, the right being most affected. The pupils were equal, and acted normally; accommodation was perfect; and there was no trace of optic neuritis. No paralysis of face or tongue. Taste, smell, and hearing apparently good. Eyesight also good; no colour-blindness. In reading, her habit was to keep her face still, and to move the book horizontally in front of her eye, so as to bring each word of a line successively into the line of vision. It was observed at this time, as it had been when she was an out-patient, that, though she appeared to have no voluntary power over the eyeballs, they occasionally executed involuntary movements. Her appetite was good, her bowels regular, her urine normal.

About the end of February, it was noted that her arm and leg had become somewhat weaker, that the arm occasionally trembled, and that she protruded her tongue towards the right.

On March 6th she began to have choreic movements of the left arm and leg. These increased rapidly during the next few days, and soon involved the muscles of the head and neck, and of expression, but did not extend to the right arm or leg. The tongue was protruded in characteristic choreic fashion, but pointed now strongly to the right, and, on being withdrawn, its point swept round to the left angle of the mouth, before it completely disappeared between the lips. It was now stated that she had had chorea when she was ten years old. And it may be added that her present attack of chorea followed on the admission of a

case of chorea into an adjoining bed. The attack was not a severe one, and had subsided by the 19th of March. No cardiac complication was discovered.

On the evening of March 9th she was sick for the first time since admission; and the next morning her headache was unusually severe. On the night of the 12th she, for the first time, had a fit, which lasted for about eight minutes. It began with sighing and crying, and rigidity of right arm and leg, the left arm and leg presenting choreic movements. She was insensible for five minutes. She did not bite her tongue, or pass her water into the bed, nor did she become livid. She had very intense headache afterwards, and scarcely slept all night. On the 18th she was again sick, and early on the morning of the 22nd had another fit, much like the former one. During its progress the right limbs first became rigid, and subsequently the left limbs. The conjunctivæ were found to be insensible.

From this time forwards the sickness became frequent; the fits recurred at intervals, varying from a few days to a fortnight; the headache grew very severe, especially in connection with the attacks of vomiting and the fits; and she had increased giddiness. Indeed from about the middle of April she was unable to stand or walk without assistance, and consequently had to remain in bed. The sickness was independent of food, and did not as a rule interfere with her appetite. The fits for a time were exact counterparts of those above described. But before long they began to occur in groups of two or three, and to present other features of interest. While at first there was no affection of temperature before, during, or after the fits, about the middle of May, and always subsequently, the temperature would begin to rise two or three or four days before the occurrence of the fits, so that as a general rule we could foretell their occurrence. With the onset of the fits, and during their progress, the temperature would fall; until on their subsidence it was found normal or subnormal. The fits came on at various times of the day, but mostly in the evening, and sometimes while she was asleep. They were generally preceded by intense headache, giddiness and vomiting. She was quite unconscious during their progress, which varied between five minutes and half an hour; and was generally more or less violently convulsed, the convulsions being general, and involving the facial muscles. The left arm and leg often became rigid and extended, and the hand clenched. On several occasions she passed water during the fits, and once bit her tongue. After all except the very earliest fits, she remained in a semi-comatose condition for twelve or twenty-four hours, or longer, during which time her pulse often rose to 130 or 140; she was apt to be restless, to pull

at her hair, to moan, and to cry out in low tones, "Nurse, dear," "nurse," "quick," "oh, my head!" "mother," &c. The left arm and leg on several occasions remained rigid for some time after a fit.

After a group of fits on the 4th of May it was observed that she had lost power wholly in the right arm, and almost wholly in the right leg. The limbs were rigid, extended, and the fingers clenched. These limbs also were slightly tremulous when lifted from the bed. These phenomena persisted with little change.

There was little subsequent change in her condition. The following was her state when she was discharged on the 2nd of February. She was still a fairly healthy-looking and plump girl; and cheerful and sensible when free from pain and fits. The muscles of her eyeballs were affected, as they had been all along; and she had a persistent double downward and inward squint. The ptosis was less marked than on admission; and occasionally still the eyeballs would move apparently independently of her will. She read, as she had done at first, by moving her book, and not her head or eyes. The pupils were equal and active. There was no loss of accommodation. No affection of the fundus had arisen. She had no colour-blindness. The tongue was still protruded strongly to the right, and its tip swept from right to left on being withdrawn. The right arm did not respond to voluntary impulses; and it remained more or less rigid and extended with the hand clenched. The right leg had perhaps undergone some slight improvement. The condition of the arm and leg was maintained during sleep. There were always exaggerated tendon-reflexes in both lower extremities, and occasionally ankle-clonus could be obtained, more especially on the right side. The impairment of sensation persisted on the right side; and continued chiefly deficient on the right side of the neck, and upper part of the same side of the chest, behind the right ear, and in the distribution of the right ulnar nerve. It was discovered also that the right side of the tongue was insensible to tactile impressions, and that the right half of the soft palate and the epiglottis were in the same condition. There was impaired sensibility also over the whole of the larynx. Smell and taste were defective on the right side. She still remained liable to fits; was rarely free from headache, which was often exceedingly intense; was frequently sick; complained of giddiness; and remained confined to bed. There was no wasting of muscles, no loss of power over the emunctories. The catamenia appeared for the first time while she was under treatment.

There are several points in the case that call for more detailed consideration than has yet been given. *

(1.) The fits appeared to me to be true epileptic fits; but there

were features about them which gave them a special interest. The first fit occurred, as has been stated, late in the evening of March 12th. It began with sobbing; was attended with rigidity, convulsions, and insensibility; lasted a few minutes; and was succeeded by intense headache. This fit came on while she was suffering from chorea. The succeeding few fits were of the same character; but it is said of one or two of them that, though insensible, she sighed during their whole continuance; and the later of them were succeeded not only by intense headache, but by a semi-comatose state, lasting for some hours, in which she was constantly moaning, and making low ejaculations, mainly complaints as to her head, and appeals to her mother and the nurse.

On the night of May 12th she had a series of three fits, and it was noticed (and I believe occurred) for the first time that the temperature (which had previously been normal) gradually rose for six-and-thirty hours previous to the fits, and fell rapidly to the normal after them. This phenomenon attended all subsequent outbreaks; and for the most part we were enabled henceforth to predict the supervention of fits, two, three, or even four days before they actually occurred. The temperature began to rise at a time when she was feeling fairly well; but its increase was always associated with increasing cephalalgia, giddiness and sickness, and general sense of illness. At the time of its highest elevation the explosion of fits occurred. During their continuance the temperature tended to fall; and as they subsided the fall was rapid. And for the most part it remained normal, or subnormal, during the day or so of semi-consciousness which always supervened. The temperature, at the moment of the occurrence of the fits, varied considerably on different occasions. The lowest was about 101° , and the highest about 105° .

I add, by way of illustration, some selected temperature charts in connection with the fits, and some of the descriptions given of the fits.

On May 29th the temperature in the morning was normal, and she seemed fairly well. In the evening it had risen to 101.4 , and she was complaining of headache and giddiness. On the evening of the 30th it had reached 102.4 , and her symptoms were aggravated. The next morning it was 103.8 . At 8.30 that evening she had a fit, which was followed in the course of the night by two others. Her temperature was not taken during the fits, but the next morning it had fallen to 98. She remained semi-comatose until the evening. (Chart 1.)

On the morning of June 5th she was free from headache and giddiness; and her temperature was 98.4 . The temperature rose rapidly during the day, and in the evening was 104 . The next

morning it reached $104\cdot2$, and in the evening $104\cdot8$. Shortly after the last temperature was taken, that is about 8 P.M., she had a fit. A second fit occurred at midnight, and a third the following morning at 7. She remained semi-comatose till late in the afternoon of the 7th, at which time her temperature was normal. (Chart 2.)

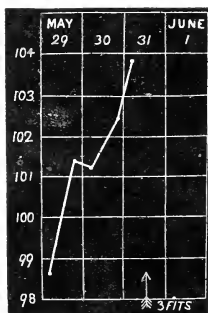


CHART 1.

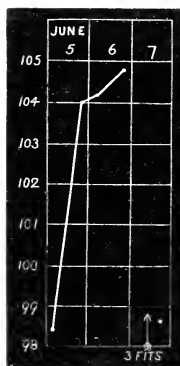


CHART 2.

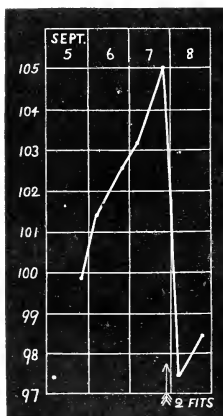


CHART 3.



CHART 4.

On the evening of Sept. 5th her temperature was $99\cdot8$, and she was complaining a good deal of headach. During the 6th her

temperature rose from 101·4 to 102·6, and her headache increased. By the evening of the 7th, the thermometer indicated 105, shortly after which time she had two fits. The next morning her temperature was 97·4, and she was semi-conscious. She had recovered about noon. (Chart 3.)

On the morning of October 13th, the temperature was normal. Later in the day it had risen to 99·6. It continued to rise irregularly during the 14th, 15th, and 16th, until in the evening of the last day it had attained 103·4. At 10 P.M. a convulsive fit came on, which was followed at midnight by two others. The temperature had fallen the next morning to 97·4. (Chart 4.)

On the morning of Dec. 15th her temperature was 98·4. From this time it rose irregularly, with increasing headache, until 5.30 P.M. on the 17th, when it had reached 101·4. At 7 P.M. the patient became unconscious, with noisy and laboured breathing. About 7·10 she was attacked with convulsions, first on the left, then on the right side. There were also twitchings of the mouth. The attack lasted 15 minutes. At 7.35 she had another convulsive fit of 15 minutes' duration; and at 8.3 another which lasted off and on for 35 minutes. During the last fit the convulsions were very violent and she bit her tongue. At 9.30 that night she was still unconscious, but her temperature had fallen to 97·4. At midnight it was 97. On the morning of the 18th her temperature had risen again to 101·2, and at noon she had a fourth fit, lasting 25 minutes, and attended with strong convulsions. At 4 P.M. her temperature was 99; at 8 P.M., 98·2. She had recovered completely but felt tired and sore, at 2 A.M. on the 19th. (Chart 5.)

On the evening of Sept. 29th her temperature was 99·8. On the evening of the 30th it had reached 101·4. The next morning, it had fallen a little. But it rose rapidly afterwards, and in the evening reached 104·4. About this time she became unconscious with rapid noisy breathing (60 in the minute); and in about 20 minutes convulsions ensued. These were clonic, and affected both sides, but mainly the left. Two other similar convulsive fits followed at short intervals. There were occasional twitchings of the right side of the face in the intervals between the fits, and after the last. The tendon-reflexes were exaggerated, and cloni could be readily obtained in both legs at these times, and also in the intervals between successive spasms. Her breathing also was very quiet and scarcely perceptible; her pulse 70. Shortly after the cessation of convulsions she became restless, and began to cry out. The temperature was normal early on the morning of Oct. 2nd; but the patient had not recovered her consciousness until late in

the evening of that day. She passed water unconsciously in this group of fits, as she had done occasionally in former attacks. (Chart 6.)

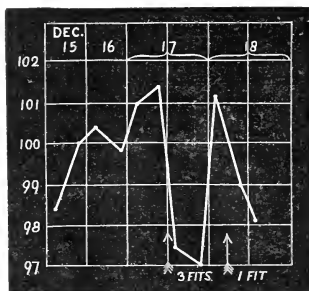


CHART 5.

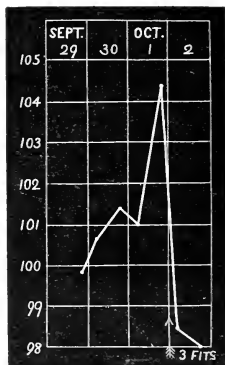


CHART 6.

The last quotation I shall make in respect to fits is from the notes taken on July 16th, and I quote these mainly because a fairly careful record of temperature was made during her epileptic state.

"Temperature has risen continuously for past five days, reaching the maximum (102.6) at 4 P.M. yesterday afternoon (Jan. 15th). A fit came on at 4.20, whilst the patient was apparently asleep. For fifteen minutes she was convulsed, throwing her legs, arms, and head about; and at the end of this period the temperature was 99.6 (fall of 3 degrees).

"For the next fifteen minutes she was perfectly quiet; breathing hardly perceptible.

"During the succeeding twenty-five minutes she lay, sometimes convulsed, sometimes trembling, with twitching of mouth, and moaning. Resp. 30, Temp. 100.2.

"She was then quiet for fifteen minutes, the temperature falling to 99.8.

"At 5.45 the respirations became quicker, and a second fit came on, commencing as before with convulsions, and passing into quiescence with occasional trembling, and twitching of mouth. Temp. 99.4.

"At 6.15 she became rather restless, and took to pulling her hair.

Temp. 98.4. She then slept for nearly two hours; during which time the temperature fell to 98." (Chart 7.)

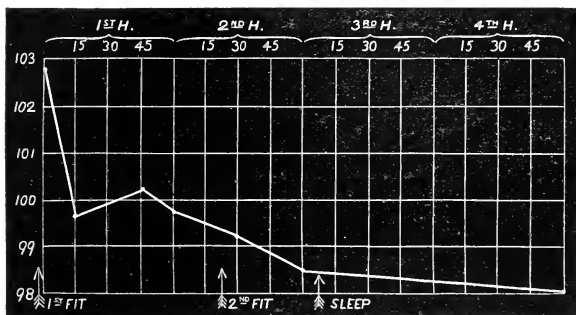


CHART 7.

(2.) For some time after the patient's admission into the hospital, her temperature (save for a rise due to an attack of tonsillitis) continued normal: and, as I have already stated, her fits during the first two months of their occurrence were not attended with any rise or change of temperature. But from this time, when the onset of fits became invariably preceded by rising body-heat, there were occasional rises of temperature, lasting even for a few days, to which fits did not succeed. The most remarkable of these began

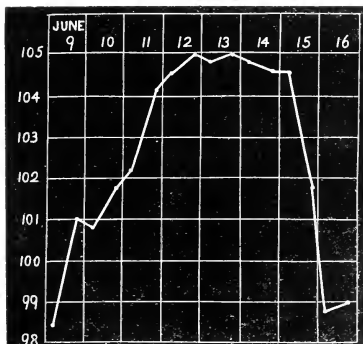


CHART 8.

on June 9th, and continued for a week. During the greater part of the time she seemed fairly well. On the night of the 12th, her headache was intense, but it was much less severe the next day. (Chart 8.)

(3.) It will be recollected that it has been stated once or twice in the foregoing notes, that, although the eyes were immovable by voluntary effort, they were occasionally seen to execute considerable lateral movements, which appeared to be involuntary. That the double inward and downward squint was not due to voluntary effort was proved by its persistence without change for many months, not only when she was awake, but when she was asleep, and by her mode of reading, which never altered. At the same time there can be no doubt of the occasional involuntary movements which, though not frequent, were witnessed on different occasions by many persons. The ptosis was never extreme, and seemed to improve of late. Mr. Nettleship, indeed, doubted whether it was true ptosis, and thought that the semi-closure of the lids might be adequately explained by the position which the eyes had assumed. The right arm and leg, after they had become paralysed, remained so. But the rigidity which was associated with the paralysis varied somewhat, and as a general rule the leg was more rigid than the arm.

(4.) The position of the pain in the head varied; at first it was mainly on the right side; subsequently it was referred usually to the occipital region; but latterly was most intense about the junction of the sagittal with the coronal suture, or over the frontal bone. The sickness was wholly unconnected with food.

(5.) The treatment adopted varied at different times. On several occasions iodide of potassium was continued for some time. Once she was treated with liquor arsenicalis. Again, bromide of potassium in large doses was had recourse to. The pain in the head was often relieved by the application of blisters, and of a few leeches. But the subcutaneous injection of the strong solution of acetate of morphia, in from four- to seven-minim doses, was on the whole most efficacious and most largely employed. The long warning given by rising temperature of the advent of fits led us on several occasions to endeavour to ward the fits off. Blisters and leeches, and the bromides of potassium and ammonium, were employed on several occasions with this object, but fruitlessly. On two occasions large doses of quinine were given and repeated, but without any apparent benefit; and on two occasions also, salicylate of soda, in twenty-grain doses every two hours, was administered as soon as there was a warning of fits, and was continued until the fit came on. On each occasion the temperature rose in spite of the remedy,

and the fits occurred as usual. Whether or not the circumstance was accidental merely, I cannot say; but it was noted that the fits following the use of the salicylates were specially severe.

CASE III.—*Ophthalmoplegia externa and interna; partial anæsthesia of head and neck and chest; epileptic fits; gastric crises; and attacks of intense dyspœa dependent on paralysis of the abductors of the vocal cords.*

ROBERT R., a labourer, 46 years old, was admitted on June 13, 1884, into St. Thomas's, from the hospital at Moorfields, where he had been under treatment for about two months. The following notes, taken at Moorfields, were given me by Mr. Lawford.

"He had had no illness for many years, and no fits. He had had venereal disease several times; the last time being two years ago, when he had 'two sores.' No evidence of constitutional syphilis.

"His present illness began three months before admission, with drooping of the left upper eyelid. This, he says, improved somewhat. Then the right lid began to droop, and gradually the ptosis became complete on that side. About a month later occipital headache and giddiness came on, with frequent vomiting. Soon after this he was in the Gravesend Infirmary for four weeks for 'inflammation of the stomach.' During this time he had severe pains in the belly, and vomiting. Never had incontinence of evacuations.

"On admission. *Right eye*.—Complete paralysis of levator palpebræ. No inward movement of globe. Slight outward movement. Slight downward movement with rotation, as if from superior oblique. Fair upward movement. Eye diverges.

"*Left eye*.—Partial ptosis; about two-thirds of cornea shown—no power of raising lid above its normal position. No outward or upward movement of globe. Very slight outward movement. Slight downward movement effected by superior oblique. Eye diverges.

Pupils dilated, without action to light. No power of accommodation. Right optic disc normal; left streaky, and sheaths of arteries and veins very visible. Media clear. No colour-blindness.

"There is double partial paralysis of fifth nerve. Motor branches apparently affected; most on right side. Masseters weak. Partial right facial palsy; cannot whistle. Tongue protruded in middle line.

"The patient has constant, though not severe, occipital head-

ache. No vomiting now. Some complaint of giddiness. Is this from the condition of his ocular muscles? No ataxy. Patellar reflexes good.

"The patient was treated with mercury. Salivation was produced, and kept up, till May 24th. Iodide of potassium was also given. But no appreciable change was observed in any of his symptoms."

On admission into St. Thomas's he was a fairly nourished, and fairly healthy-looking man, complaining of occipital headache and giddiness. The condition of his eyes was exactly that described by Mr. Lawford. He could not whistle or puff out his cheeks, and his aspect was expressionless; but he could show his gums fairly well, and he laughed symmetrically. I could not satisfy myself that there was any clear paralysis of the facial nerves. When he opened his mouth widely the chin was slightly thrown over towards the left side. This was the only phenomenon which suggested any implication of the motor branches of the fifth Tongue protruded straight. There was marked impairment of sensation all over face and front of neck; also over front of the chest and abdomen to within an inch or two of the umbilicus. There was no weakness or defect of sensation in the arms or legs, and the tendon and superficial reflexes were natural. No impairment of hearing, smell or taste was detected. His voice and articulation were perfect. His mental condition was unaffected. The abdominal and thoracic viscera appeared to be healthy. He slept well. His tongue was coated, his appetite fair. Bowels regular, urine normal.

Little or no change took place in the nervous symptoms above described during his stay in the hospital. But he presented from time to time several additional interesting phenomena, which I will describe. They were, a sense of weight at the epigastrium, which was sometimes so intense as to make him roll about in bed and cry out; epileptic convulsions; and attacks of intense dyspnoea, in which at times he seemed in immediate danger of death.

(1.) He complained pretty constantly of pain across the loins, and occasionally of pain along the spine. But often, and at irregular intervals, he suffered also from a sense of weight or oppression or constriction in the epigastrium (for the most part associated with the dorsal pain), which was often so severe as to make him groan and cry out in agony, but which he did not describe as being actual pain. This sensation was generally aggravated by breathing deeply, speaking, moving, or taking food; and consequently when at his worst he would lie in bed,

groaning, speechless, and refusing all nourishment. He felt during the attacks as if he should be suffocated, and also as if he should be relieved by vomiting. Retching and vomiting indeed were not uncommon. But relief never ensued therefrom. During the attack his breathing was for the most part slow and shallow and quiet, but not infrequently a deep inspiration occurred, which was always noisy. The pulse was quick and weak, and the temperature normal. It did not appear to me that he had true dyspeptic symptoms, or that there was any actual loss of appetite or disgust for food. The attacks here described, sometimes slight, sometimes severe, and lasting for a few hours, or for a day or two at a time, were common. But he had one or two severe attacks of a week or two's duration each, in which his sufferings were constant and most severe, in which he practically refused all food, and in the course of which he became so weak and ill, that it was feared he would sink. At these times he found considerable relief from morphia injections. Ice to the epigastrium comforted him on one or two occasions.

(2.) It was during a prolonged bout of epigastric discomfort and vomiting, that on August 11th and 12th he had four fits. These were of sudden occurrence, of short duration, and attended with absolute insensibility and slight convulsive movements of his hands. With the latter exception he lay as if he were dead. In the intervals he vomited, his respirations were 40, laboured and snoring, and his pulse about 130. Another fit occurred on the 9th of October. But there was no further recurrence.

(3.) He was liable throughout his stay in the hospital to sudden attacks of extreme dyspnœa, which lasted from a second or two to 5 or 10 minutes at a time. The first of these was observed one day while he was at lunch. He suddenly became livid in the face, struggled violently for breath, and made loud snoring inspirations. The dyspnœa subsided after a few minutes. All his other attacks were the same in quality; but often, and more especially during the latter period of his stay in the hospital, they were of littel more than momentary duration. They came on quite irregularly, sometimes in the day, sometimes at night, and while he was asleep; and were often very alarming. On the 2nd of December, Dr. Semon examined his throat with the laryngoscope, and reported that there was complete paralysis of the left abductor, and incomplete paralysis of the right. "The left vocal cord (the inner border of which is slightly excavated) stands perfectly immovable in the middle line; the right one (which appears similarly excavated) is, on attempt at deep inspiration, hardly drawn outwards to the cadaveric position, so that the chink of the glottis is always

very narrow. On attempted phonation, both cords meet completely; the right one and the right arytenoid cartilage being promptly drawn to the middle line."

(4.) During his stay in the hospital he had a few accidental complications. Shortly after admission he had some conjunctival inflammation which lasted for a few days. A month or two later his temperature rose for several days, and on one of them he coughed up about an ounce of blood, the exact source of which was not ascertained. And at the beginning of March 1885 he had a sharp and severe attack of erysipelas, commencing from the right eyelid. Excepting at these times, his temperature was always about normal.

His treatment consisted mainly in the continuance of the iodide of potassium and mercury. For his headache and other pains, cannabis indica and morphia (by subcutaneous injection) were occasionally administered; for the attack of dyspnoea, nitrite of amyl and nitro-glycerine; and as local applications to his epigastrium, ice and counter-irritants.

When he left the hospital on the 31st of March, he was still complaining of occasional catches in his breath, especially in the morning, and of some oppression at the chest; and all his paralytic phenomena remained as they have been described. But in his general health he felt better than he had done for a long time.

CASE IV.—*Ophthalmoplegia externa. Wasting Palsy.*

JOHN M., a sauce-maker, aged 53, consulted me on two or three occasions in the summer of 1884. With the exception that he had had syphilis, he had enjoyed good health up to twelve years ago. At that time he first complained of double vision. Five years later his eyelids began to droop. About four years ago, weakness and wasting of the muscles of the left shoulder and upper arm came on. Subsequently the right arm and the thighs became similarly affected. He states that there has been some variation in the condition of his eyes; and that there have been times when he has been able to raise his lids fairly well. Latterly he has suffered from gout.

The patient presents almost complete ophthalmoplegia externa. He has very slight power over his upper eyelids, which cover half the corneæ and pupils. His eyeballs are prominent, and can only be moved very slightly (in an arc of about one-sixth inch), inwards and outwards; not at all in any other direction. There is no internal paralysis of the eyes, no impairment of sight, and no

affection of either fundus. The right masseter is somewhat wasted, and the lower jaw is thrown over to that side when he opens his mouth widely. There is marked weakness and wasting of both shoulders and upper arms, and slight wasting of the forearms. The thighs are small; but, excepting for gout in his feet, he seems to walk fairly well.

Patellar tendon-reflexes normal; no numbness of feet or hands; no lightning pains, or indeed any symptoms of locomotor ataxy.

CASE V.—*Ophthalmoplegia externa and interna. Locomotor Ataxy, attended with gastric and rectal distress, and a peculiar cough.*

A musician, between 30 and 40 years of age, was admitted under my care in 1880. At that time he was suffering from obscure symptoms, the meaning of which I failed to interpret. He was exceedingly nervous and irritable, and gave me the impression that he was hysterical. Whether I did not trouble myself to investigate his case thoroughly, or whether I did investigate and could discover nothing to explain his symptoms, I cannot recollect. The case, however, made little impression on me, and I should have forgotten all about it, but for its subsequent history. He called upon me at my own house, a little later in the year, complaining of "tightness in his leg;" but he walked without difficulty and well; and I still failed to discover what was the matter with him.

The next time I saw him was in April 1883. On this occasion he had a marked ataxic gait, and complained of numbness in his feet, and occasional slight lightning pains in his legs. His eyes also had become affected. There was partial ptosis on the left side. The pupils were dilated, and acted neither to light nor to accommodation, and he had lost the power of adjusting his sight to near objects. But in other respects his sight was good. He could move his eyes readily in any direction, and there was no affection of the fundus of either of them. He complained also of frequent pain between the shoulders, and sickness, and of almost constant severe pain in the lower part of the back. He was still nervous and hysterical in manner. His appetite was good.

I saw him again in June 1884. He had then become much worse. The movements of his legs were strongly ataxic, and he could not walk or stand without assistance. The tendon-reflexes were abolished. He had no lightning pains, but he complained of occasional pains in the ankles, as if they were being scratched. The pupils were dilated, but unequal, and acted neither to light nor to

accommodation. He said he could not focus his eyes. The mobility of the eyeballs was now much impaired; he could not move either to left beyond middle line. Movements to right imperfect. Elevation of both equal, but slight. In trying to look down, the right moved less than the left. No colour-blindness. He had a fair appetite, but ate little, for fear of aggravating the pain in his chest, which was always present, more or less. He complained mainly, however, of pain in the rectum and lower part of the back, which was constant, but liable to exacerbations, and the source of intense misery to him. Bowels confined. He had suffered for some time from a cough (of which I witnessed several attacks), consisting of a series of ineffectual expiratory shocks, followed by a noisy, somewhat crowing inspiration. Voice natural; no dyspnoea; no dysphagia. It is not improbable that the peculiar cough may have been connected with some paralytic affection of the vocal cords, but unfortunately I did not investigate this point.

ADDENDUM TO ACCOUNT OF AUTOPSY OF MARION H.—There are pale yellow patches, not differing in consistence from the surrounding brain-substance, scattered here and there in both grey and white matter. They appear to be local areas of anaemia, occasionally seen in otherwise normal brains.

The patches are most evident, (1) in the cortex of the third left transverse frontal convolution; (2) in the outer part of the anterior extremity of the left lenticular nucleus; (3) in the internal capsule and adjoining part of the lenticular nucleus posteriorly. The right anterior crural and median nerves healthy. Cervical sympathetic also healthy.

Microscopical Examination.—Fifty sections from various parts were examined. The cortex and underlying white matter of the left third frontal convolution were healthy, as were also the motor and sensory portions of the internal capsule on the same side, the lenticular nucleus, the claustrum and the island of Reil.

Nothing abnormal was detected in the corpora quadrigemina. Owing to an error, the condition of the nuclei of the third nerves was not made out.

The fibres of the sixth nerves and their nuclei were perfectly healthy. The nuclei of the facial nerves, the spinal accessory, the glosso-pharyngeal, the vagus and the hypoglossal were also normal.

There was no sclerosis in the pyramidal tract, pons, or medulla oblongata.

The right median nerve and the superior cervical ganglia of the sympathetic were normal.

ON SOME CENTRAL AFFECTIONS OF VISION.

BY W. J. DODDS, M.D., D.SC.

(Continued from p. 39.)

LOCALISATION OF THE VISUAL REFLEXES.

THE first part of this paper contained an enumeration of the visual reflexes. We have now to enquire, What is the localisation of these reflexes, what are the sensori-motor tracts along which they pass? We shall endeavour to answer this question as definitely as our present knowledge will allow. Our answer will unfortunately be incomplete, and present many lacunæ; but we find consolation in remembering that something is gained even by a formal enunciation of things we do not know. A clearer objective is presented for future investigators to make for.

(1.) *The Light Reflexes.*—We have seen that three separate reflex actions are included under this head, viz. the contraction of the pupil to light, the dilatation of the pupil on removal from a bright to a dull light, and the general motor effects produced by a strong glare of light.

(a.) What portions of the brain must be intact that the pupil may contract to light? It is commonly believed that the reflex travels along the optic nerve and tract to the anterior corpora quadrigemina, thence to the underlying nucleus of the 3rd nerve on the floor of the aqueduct of Sylvius, and thence by the root of the 3rd nerve to the sphincter iridis. The nuclei of the 3rd nerves are united by commissure, so that the nerve-current generated by the illumination of one eye stimulates the nuclei of both third nerves, and causes contraction of both pupils. An attempt has recently been made to upset this view of the localisation of the reflex. Bechtereff,¹

¹ 'London Med. Rec.' 1884, p. 57.

as the result of experiments on dogs and birds, states that the course pursued is from retina to chiasma, thence to the grey matter surrounding the 3rd ventricle, and so to the nuclei of the 3rd nerve; there are, he says, no reflex pupil-constricting fibres either in the optic tracts, corpora geniculata, or corpora quadrigemina. Let us see if any light is thrown on this subject by the facts of pathology. What effect has destruction of the optic nerve on the reflex contraction of the pupil to light? Lawson¹ gives a case in which the optic nerve was diagnosed as wounded from a stab with a knife, and the globe was uninjured; there was absolute blindness, and the pupil was uninfluenced by light. In cases of complete amaurosis from atrophy of the optic disc, the reflex contraction of the pupil is usually very faint, and it may be altogether absent; in the latter case, however, a slight reflex can generally be elicited after a time. Hutchinson² indeed remarks that he doubts whether in almost any case, however great the degree of blindness, the pupils become permanently motionless, provided the motor nerves of the iris remain intact, and he thinks that the 5th nerve may take upon itself the duties of excitomotor. Another explanation would be that all the fibres of the optic nerve are not destroyed, and that enough remain to serve as an afferent track for the reflex, though not enough to relieve the amaurosis.

What effect has destruction of the optic tract on the contraction of the pupil to light? Wilbrand³ states, though without citing cases, that in amaurosis from disease of both optic tracts the pupils are dilated and uninfluenced by light. The same author gives a table of seven cases of lesion of one tract, but in only two of them is any mention made of the condition of this reflex; in both the reflex was present, but was sluggish. The cases, however, prove little, for the problem is complicated by the existence of a partial decussation in the chiasma, so that each eye is represented in both optic tracts; and even if one optic tract is destroyed, the light stimulus is still able to travel along the other optic tract. Bechtereff's view is opposed by several cases⁴ of atrophy and extreme

¹ 'Lancet,' 1875, i. p. 13.

² 'BRAIN,' Vol. I. p. 6.

³ 'Ophthalm. Beiträge,' 1884, p. 57.

⁴ See Wilbrand, 'Hemianopsie,' p. 91.

thinning of the tuber cinereum, in which the pupil reflex was normal and all eye symptoms absent.

Tracing the optic fibres a step further back, we ask, What effect has lesion of the anterior corpora quadrigemina on the reflex under consideration? Numerous cases could be cited where lesion of the corpora quadrigemina, especially of its anterior tubercles, was coincident with absence of the reflex; but when we look for a case sufficiently uncomplicated to satisfy scientific precision, we have to confess that we look in vain. Localised lesions of the corpora quadrigemina are amongst the rarest of brain lesions. Bristowe¹ gives a case in which a tubercular tumour, marble-sized, occupied the corpora quadrigemina, and was jammed between their upper surface and the aqueduct of Sylvius; the pupils reacted badly to light about three weeks before death, but normally during the previous three months. The abolition of the reflex, so common in locomotor ataxia, is sometimes attributed to disease of the radiating fibres that pass from the ganglia of the anterior corpora quadrigemina to the oculo-motor nucleus, but we know of no post-mortem confirmation of this hypothesis.

Lesions of the nucleus of the oculo-motor nerve and its nerve-fibres abolish the reflex contraction of the pupil to light. The nucleus and its nerve are included in transverse sections that embrace the lowest part of the crura cerebri and the uppermost part of the pons.

We have now discussed the question, What parts of the brain must be intact that the pupil may contract to light? We have seen that there is still an element of doubt as to whether a very slight contraction of the pupil may not be evoked, even though the optic nerve is destroyed, and it would be desirable to have additional clinico-pathological evidence that destruction of the optic tract causes loss of contraction. But in the main there seems no reason to doubt the accuracy of the generally received localisation. It will have been remarked that the reflex is difficult to abolish, except at the afferent or efferent termination of the chain, owing to the presence of optic fibres from each eye in both

¹ 'BRAIN,' Vol. VI p. 173.

optic tracts, and to the fact, that the nuclei of the 3rd nerves are united commissurally. In unilateral affections then, if the contraction of the pupil reflex is not evocable in one eye, the lesion is probably in the optic nerve or the oculo-motor; if it is evocable by stimulating the other eye, the lesion is not in the oculo-motor, but probably in the optic nerve.

(b.) The pupil dilates on removal from a bright to a dull light. This reflex, as is well known, is due to the tonic action of the radiating fibres of the iris. It strictly is not a visual, but a sensory reflex. The efferent fibres supplying the dilator iridis may be traced to two sources: on the one hand, by way of the cervical sympathetic, to the upper dorsal and lower cervical region of the cord (*centrum cilio-spinale inf.*), and thence to the medulla oblongata; on the other hand, to the 5th nerve, the further course of this set of fibres being unknown. Destructive lesions in the inferior cilio-spinal region are known to produce myosis from paralysis of the dilator iridis, but they also produce at times mydriasis from predominance of the irritative phenomena. Paralytic myosis is very rarely recorded in lesions of the medulla oblongata, but we notice that Baerwinkel¹ gives it as a symptom in sclerosis of the medulla oblongata. Unilateral myosis of long duration is stated by Nothnagel² to be sometimes observed in diseases of the pons; whether in relation to the just named fibres of the fifth, we cannot say.

(c.) The motor phenomena produced by the sensation of light as a pleasurable or painful feeling. We know of no cases that enable us to determine the localisation of this reflex. The movements of uneasiness that are produced by the shining of an extremely bright light into the eye, and that are developed though there is blindness, are due, we should surmise, to irradiation in the region of the grey matter at the base of the brain underlying the anterior extremity of the 3rd ventricle. The grey matter here is known to be one of the centres of origin of the optic fibres. If this surmise is correct, we should find this reflex present, notwithstanding complete destruction of both optic tracts.

¹ See Eulenburg and Guttman, 'Journ. Ment. Sci.,' 1878, p. 383.

² 'Topische Diagn. der Gehirnkrankheiten,' p. 593.

(2.) *Objects fixed and followed by the eyes.*—(a.) The retino-ciliary, or accommodation reflex. The afferent nerve of this reflex is the optic, the centre is in the anterior corpora quadrigemina, probably, and in the nucleus of the 3rd nerve, and the efferent nerve is the 3rd nerve. Accommodation by voluntary effort is possible without the formation of an image on the retina, but for all practical purposes we may say that a retinal image is required, and vision. What then is the course traversed by the reflex from the optic nerve to the nucleus of the 3rd nerve? It is probably through the anterior corpora quadrigemina; but whether this suffices, or whether the agency of the higher ganglia is needed, is a point that cannot be settled at present. Central affections of this reflex are rare. The best known instance is as one of the sequelæ of diphtheria, when there is paralysis of accommodation, or cycloplegia, as it is conveniently called; and there is a doubt whether, after all, the lesion is really central or peripheral.¹ Cases of accommodative asthenopia, uncomplicated by errors of refraction, are probably due to a functional implication of the central track of this reflex. As regards the exact location of the centre for the ciliary muscle in the long column of nerve-cells that forms the 3rd nucleus, we cannot do better than quote from Gowers's² recent book, "that the combined teaching of experiment and of clinical observation, shows that there are three centres in this column of nerve cells, distinct at least in function and in pathological liability. The most forward of these is the centre for the ciliary muscle (accommodation); the second is the centre for the light reflex of the iris; the third, which occupies the greater part of the nucleus, is the centre for the external muscles supplied by the third nerve." Contraction of the ciliary muscle is generally accompanied by a contraction of the pupil.

(b) The retino-ocular reflexes. The afferent fibres are in the optic nerve and tract, the centre in the anterior corpora quadrigemina, and the grey matter on the floor of the aqueduct of Sylvius and fourth ventricle, reaching from the most

¹ Meyer, 'BRAIN,' Vol. IV, p. 561.

² 'Diseases of the Brain,' p. 29.

anterior portion of the anterior corpora quadrigemina to the most posterior portion of the pons; and the efferent fibres are in the roots of the 3rd, 4th, and 6th nerves. The afferent¹ and efferent tracts call for little notice, and we shall pass on at once to the more obscure subject of the central paths of these reflexes.

The sensory centre lies in the anterior corpora quadrigemina, the motor in the grey matter just described. Radiating fibres are seen to pass from the former in the direction of the latter, and they, in all probability, form the connection between the sensory and the motor centres. Other pathways have been described; Quioe,² for example, thinks that the corpora quadrigemina are connected with the 6th nerve by means of the fasciculus pedunculus transversus of Gudden. Our knowledge of the interconnection of the motor nuclei themselves is chiefly due to the labours of Duval, Laborde, Graux, and others. Most authors agree that the fibres of the posterior longitudinal fasciculus form a bond of union between the nuclei; but there is still considerable difference as to the details of the connection, and the bilateral grouping of the nuclei is uncertain. Duval³ describes a connection between the nucleus of the 6th nerve of one side and the 4th nerve of the same side, the 3rd nerve of the opposite side. This tract would therefore subserve the associated or conjugate movements of the eyes, and would explain the associate action of the right externus and left internus muscles. Roller,⁴ too, affirms that this fasciculus receives fibres from the 6th and 4th nuclei, but he has not yet been able to trace its connection with the 3rd.

We have glanced at the anatomical side of the question, let us now see if pathology can make any addition to our knowledge. And first let us consider the associated lateral action of the eyes. Foville,⁵ as early as 1858, held that a

¹ Roller ('Archiv f. Psych.' xi. p. 260) has suggested that a reflex route between retina and eye-muscles may be by way of the optic nerve, nucleus amygdaliformis, post. long. fasciculus, and so to the nuclei of the eye-muscles.

² 'Archives de Neurol.,' vi. p. 112.

³ Duval and Laborde, 'Archives de Neurol.,' i. 124, and 'Journ. de l'Anat. et de la Physiol.,' 1880.

⁴ 'Archiv f. Psych.,' xi. p. 260.

⁵ 'Bull. Soc. Anat.,' 1858.

centre for conjugate lateral movements of the eye was situated in each abducens nucleus, or in its immediate neighbourhood. This localisation is supported by Féréol's case,¹ in which a tubercle implicating the 6th nucleus produced paralysis of the externus of the right eye and internus of the left, power of convergence being retained, and the movements of the left eye being free in monocular vision. Doubt has, however, recently been thrown on this view, and Senator, in an interesting paper,² cites cases in which there was lateral paralysis without affection of the 6th nucleus. As regards one class of these cases, the holders of Foville's view save their theory by suggesting that there was lesion of the voluntary motor tract leading to the 6th nucleus, or by looking on the centre as stretching the whole way between 3rd and 6th nuclei; and there is no doubt justice in the plea. But there are other cases³ in which neither the 6th nucleus nor the voluntary motor tracts were implicated, though there was still paralysis of lateral movements. We can therefore at present do no more than accept Senator's indefinite conclusions, that in lesions of the pons, with associated lateral paralysis of the eyes, the 6th nucleus may or may not be implicated; there may either be lesion above the 6th nucleus affecting the voluntary tract, or peripherally to it, affecting the root fibres of the 6th near the nucleus.

Next as to movements of convergence. The movements of convergence are distinct from the lateral movements. There may be paralysis of the latter with retention of the former; in other words, the conjugate action of the internus of one eye and the externus of the other may be paralysed, while the conjugate action of the two interni is retained. This would point to a separate innervation of the internus, according as it is used in associated lateral movements, or in convergence, and there is reason to believe⁴ that the centre for convergence lies in front of the centre for the lateral movements. Parinaud⁵ argues that the cerebellum is the co-ordinating centre for movements of convergence. It is, he says, from these

¹ 'Union Méd.,' 1873.

² 'Archiv f. Psych.,' xiv. p. 643.

³ Ibid. p. 654.

⁴ Ibid. p. 658.

⁵ 'Archives de Neurol.,' v. p. 145.

movements that we derive our notions of the third dimension, which are closely related to the notions of space, given us by the vestibular branch of the auditory nerve, and it is only proper, therefore, that both should have their centre in the cerebellum.

(*e*) Convergence of the eyes is attended by contraction of the pupil; but we know of no cases that throw light on this phenomenon pathologically.

We have up to this point regarded the retino-ocular reflexes as meso-cephalic, and independent of higher cerebral influences. That such influences play a very prominent part in the movement of the eyes, no one can doubt; they occur whenever the eyes are moved voluntarily, or are directed toward some object in the projected visual field of the imagination. But the question at present before us is—Can an object be seen and followed by the eyes without the action of the optic thalamus, or the cerebral hemispheres? There can be no doubt as to the answer to this question, in the case of some of the lower animals. Flourens showed that, after the cerebral hemispheres were removed from a pigeon, the bird still followed with its eyes the movements of a lighted object. In the case of man, we have in the main to be content with such unsatisfactory conclusions as can be founded on analogy. Little weight can be attached to an isolated observation of Dor's,¹ where the left eye is said to have seen nothing, and yet followed the movements of a finger in every direction. Witkowski² states that in sleep the movements of the two eyes are extremely slow; they are inco-ordinate, each eye appearing to move on its own account; they are neither influenced by light nor by sensory influences, and the pupil takes no part in them, even though the eye converges. In conditions of stupor, such, for example, as often occur in the last days of life, the movements are co-ordinated, but the eyes move from side to side without taking any notice of objects, and the movements are accelerated by light. These facts would suggest that in man the action of the cerebrum is necessary for the retino-ocular reflexes, for there is some reason to believe

¹ Wilbrand, 'Hemianopsie,' xi. p. 120.

² 'Archiv f. Psych.,' xi. p. 507.

that it is chiefly the cerebrum that is affected in sleep and the lighter forms of stupor. But, in truth, such conclusions should be received with the greatest reserve.

Practically, if this reflex is absent from lesion above the corpora quadrigemina, it will either be from destruction of the afferent tract (blindness), or of the voluntary motor tract for the muscles of the eye. The consideration of the former is taken up later; as regards the latter, it is extremely rare, and the rarer the more centrally we go. In these cases Senator's test may help us; the paralysis can be overcome in a measure by voluntary effort, since the nerve-nuclei can be reached from the other side of the brain.

The various reflexes we have just been considering are all grouped together and co-ordinated, in the act of looking at an object, and are further associated with certain movements of the head and neck. A case of Gowers¹ is a good example of impairment of these reflexes: if the patient was told to look at an object at an angle from that at which he was looking, of say 45°, his head was instantly turned toward the object; but the eyes were kept fixed on the first object, and they slowly followed the movement of the head until they were again in mid-position and fixed on the required point. What is the localisation of this complex group of reflexes? There is a difference of opinion whether it is in the medulla and pons, or in the cerebellum. Let us see what evidence can be adduced in favour of the latter.

Anatomy has already mapped out a route by which the afferent impulses would travel, Stilling² having traced one of the roots of the optic nerve from the superior corpora quadrigemina to the velum medullæ, and thence by the superior cerebellar crus to the cerebellum. Ferrier³ has shown that electrical stimulation of the cerebellum causes associated movements of both eyes in various directions with contraction of the pupil, movements of head and eyes, and also of the limbs on the same side of the body; and Duval and Laborde⁴ conclude from their experiments that the general co-ordinating centre for the ocular movements is seated in the cerebellum

¹ 'BRAIN,' Vol. II. p. 39.

³ 'Functions of the Brain,' p. 99.

² 'Archiv f. Psych.,' xi. 274.

⁴ Op. cit.

more especially in the inferior vermiform process. Clinical observers agree that lesions of the cerebellum are not unfrequently attended by ocular motor symptoms, such as strabismus, uncertain movements of head and eyes, nystagmus, difficulty in fixation, etc., though they also agree that none of these are pathognomonic of cerebellar affections. Nothnagel¹ indeed states that strabismus convergens, though the most frequent of the ocular motor troubles in cerebellar disease, is yet comparatively rare and never occurs in apoplectic lesions. The two commonest symptoms of cerebellar disease are muscular inco-ordination and vertigo. Vertigo may undoubtedly be due to disturbances in the sphere of the optic fibres; but it is so complex a symptom, and has so many causes, that we cannot base any argument on it here. The inco-ordination has only rarely been observed in the movements of the eyes, though perhaps the reason is, that observation has not been sufficiently directed to this point. On the whole, it cannot be said that the evidence of the localisation of general ocular co-ordination in the cerebellum is at all satisfactory or convincing.

The co-ordinating centre for the muscles of the eyes and head and neck is located by other authors in the pons and medulla. To this view also anatomy lends its shield. One indeed is sometimes tempted to say that it would be difficult to propound a theory to which our too-obliging anatomy would not lend its support. Spitzka² has described strands as passing from the anterior corpora quadrigemina downwards to the cervical cord, and so connecting a centre for visual impressions with centres that govern movements of head and neck. Roller³ again describes the optic nerve as giving fibres to the band of Reil, and so passing to the ground-bundle of the anterior columns of the cord. Experimental physiology has shown that unilateral injury of the medulla oblongata from the calamus scriptorius to the tuberculum acusticum causes strabismus and nystagmus, with constrained movements and posturings of the head and body, and it is argued that where eye-symptoms occur in cerebellar disease, they are not due to

¹ Op. cit., p. 72.

² 'New York Med. Rec.,' 1880.

³ 'Allegem. Zeits. f. Psych.,' xxxviii., and 'Archives de Neurol.,' vi. 94

it, but to its effect on the medulla and pons. Thus Bernhardt explains the convergent squint in cerebellar lesions by compression of the abducens at the base. The localisation of these reflexes must remain at present unsettled. It is not unlikely, we think, that the superior cerebellar crus, middle vermiform process, middle cerebellar crus, and pons, all have a part in them as representing the sensori motor track of the reflex.

Following the order we observed in the first part of this paper, we shall next consider the localisation of the lesion in cases of hemianopsia, amblyopia, and amaurosis.

Hemianopsia.—Lateral homonymous hemianopsia, and it is of this only that we shall speak, is found in unilateral lesions of the optic tract, the pulvinar or posterior portion of the optic thalamus, the optic radiations of Gratiolet, and the cortex of the occipital lobe. There is no evidence of a unilateral lesion of the anterior corpora quadrigemina causing hemianopsia; the point, however, needs further investigation. That lesions of the *optic tract* produce hemianopsia has long been admitted, and there is no need for us to cite illustrative cases. We shall merely refer the reader to Wilbrand's able and comprehensive works¹ for a list of cases, at the same time acknowledging our indebtedness to these works in preparing this part of our subject. The recorded cases of hemianopsia from disease of the *pulvinar* are few in number. The most convincing is a case of Hughlings-Jackson's² of left hemianopsia with left hemiplegia and hemianæsthesia, the autopsy disclosing as the sole lesion a focus of softening limited to the posterior half of the right optic thalamus (see also Dreschfeld's case³). Additional observations are necessary before we can form just conclusions as to the connection between the posterior portion of the optic thalamus and hemianopsia. As examples of hemianopsia from lesion of Gratiolet's *optic radiations*, we may mention a case by Richter,⁴ of hæmorrhage into the centrum ovale

¹ 'Hemianopsie,' and 'Ophthal. Beitr. zur Diagn. der Gehirnkrankheiten.'

² 'Lancet,' 1874.

³ 'BRAIN,' vol. iv., p. 549.

⁴ Wilbrand's 'Ophthalm. Beitr.,' p. 62.

external to the posterior cornu of the lateral ventricle; a case by Wernicke and Hahn's,¹ of abscess extending from the most posterior part of the occipital and parietal lobes, and destroying the white matter outside and above the posterior cornu; and a case by Dmitrowski and Leberden,² of hæmorrhage into the centrum ovale, implicating the greater part of the corona radiata, and affecting especially the white matter underlying the two upper temporo-sphenoidal convolutions. Of these cases, and they are the best we know, Richter's is the only one that affords a hint as to the localisation of the tract, destruction of which causes hemianopsia, so that here again we have to suspend judgment until additional data are forthcoming.

Cases of hemianopsia from lesion of the cortex or subjacent medulla of the *occipital convolutions* are comparatively numerous. Wilbrand gives a table of fifteen cases, and for others we would refer the reader to Starr's³ paper. We shall cite the most important. Haab⁴ reports a case of complete lateral left hemianopsia lasting a year, in which there was a focus of softening at the tip of the right occipital lobe, extending on the mesial aspect of the lobe for a distance of 6 cm. forward, and implicating the parts round the sulcus hippocampi; the lesion was confined to the cortex. The same author gives another case of left hemianopsia, in which a small tumour was found in the sulcus hippocampi on the mesial aspect of the tip of the right occipital lobe; the tumour was surrounded by a zone of yellow softening. Féré⁵ records a case of right hemianopsia with superficial softening of the greater part of the cuneus, and a small portion of the adjoining 2nd temporo-occipital convolution; the grey commissure of the 3rd ventricle was absent. Then we have Curschmann's case⁶ of left hemianopsia with a large softening in the right occipital lobe, reaching to the surface, and involving chiefly the tip and upper part of this lobe; Baumgarten's⁷ complete and absolute

¹ Wilbrand's 'Ophthalm. Beitr.,' p. 62.

² Ibid.

³ 'American Journ. Med. Sci.,' 1884.

⁴ See Wilbrand's 'Ophthalm. Beitr.,' p. 66.

⁵ 'Archives de Neurol.,' ix. p. 222.

⁶ See Wilbrand's 'Ophthalm. Beitr.,' p. 68.

⁷ Ibid.

left hemianopsia, with apoplectic cyst, walnut-sized, in the substance of the right occipital lobe, separated below from the posterior cornu by a thin layer of intact medulla, and bounded above by the three occipital convolutions, all in a state of yellow softening; there was a small softening in the roof of the left anterior cornu, and a small cicatrix in the centre of the right optic thalamus; and Marchand's,¹ complete left hemianopsia, with a necrosed patch, the size of a hazelnut, at the tip of the right occipital lobe, the morbid process at the same time affecting the adjoining convolutions on the convex surface of the lobe. On these six cases rests the burden of proof that lesion of the occipital convolutions can cause hemianopsia, and in our opinion they establish it. A more precise localisation is scarcely feasible at present.

Is there evidence that lesion of any other portions of the cerebral hemispheres will produce hemianopsia? Lesion of the angular gyrus, for example? We know of no case that warrants such a conclusion. In all the cases of cerebral hemianopsia with which we are acquainted, with but one exception, the occipital lobe was implicated, or the optic radiations destroyed close to the optic thalamus. The exception is a case of Huguenin's², in which there was necrosis of the posterior end of Broca's convolution, lower portions of the central convolutions, anterior part of the supramarginal convolution, upper portions of the insula, the claustrum, the outer segment of the lenticular nucleus. Nothnagel suggests that the hemianopsia may have been caused by the extension inwards of the disease from the supramarginal gyrus to the optic radiations as they issue from the internal capsule. Be this as it may, we cannot, in face of the facts adduced, accept Féré's view that the cerebral centre for bilateral vision is in the neighbourhood of the cortical motor centres, between the fissure of Rolando and the angular gyrus, and near the fissure of Sylvius. Space will not permit of our noticing the differential diagnosis of the hemianopsias produced by lesion of the different parts of the optic nerve tracts; an excellent *résumé* of this will be found in Wilbrand's book.³

¹ Wilbrand's 'Ophthalm. Beitr.,' p. 68. ² Nothnagel, *op cit.*, p. 398.

³ 'Ophthalm. Beiträge.'

Amblyopia.—Amblyopia is essentially a diminution of the visual acuity that cannot be accounted for by any ocular changes. But side by side with this impaired acuity, indeed as a consequence of it, there is generally limitation of the fields of vision for white light and for the different colours. These three factors enter into every case of amblyopia, and their condition should always be noted, as well as the degree of affection of each eye.

Amblyopia may be caused by disease of the optic nerves. In cases of atrophy of the optic nerve, even before implication of the disc, there may be amblyopia. The visual fields vary in different cases. In one class, as in tabetic amblyopia, the limitation of the field of vision for white is peripheral, though it may be segmental or even hemianopic; there is diminished sensibility to colour, and both eyes are generally affected, though not to the same degree at the same time. In another class, as in tobacco amblyopia, the fields for white light and for colours are diminished at the centre, and both eyes are equally affected; Gowers states that there is frequently also peripheral dimness. It is important to remember in what diverse conditions atrophy of the optic nerve occurs, and how frequent amblyopia from this cause is.

We do not know of any cases of amblyopia (excluding of course hemianopic defects) from disease of the optic tracts, or corpora quadrigemina, or optic thalamus.

Disease of the posterior third of the posterior limb of the internal capsule is very widely believed to produce amblyopia. The amblyopia is characterised by diminution of visual acuity, peripheral contraction of the field of vision, and impaired sensibility to colour; these characters being observed in both eyes, but much the more pronouncedly in the eye on the side opposite the seat of lesion. Let us examine the evidence on which this view is founded. Féré,¹ a strong supporter of it, has only been able to collect seven cases of amblyopia with ascertained lesion of the posterior portion of the internal capsule. We shall cite the strongest of these. In Müller's case there was a pea-sized focus of softening at the posterior point of the external segment of the lenticular nucleus, and in the adjacent white matter; the

¹ 'Amblyopie croisée et Hémianopsie,' 1882.

symptoms affected the right side only, and comprised a pronounced amblyopia of right eye, complete sensory hemianæsthesia, with unilateral affection of the special senses, and a slight hemiparesis. We would remark in this case on the striking disproportion between the extent of the lesion and the observed symptoms; it is so great, indeed, that we cannot accept the lesion as the sole cause of the symptoms. It is noticeable too that this is the only case in which the amblyopia was not bilateral; the left eye is said to have been quite normal. In Pitres's case there was lesion of the right internal capsule at the junction of its posterior fourth with its anterior three-fourths, and also of the right optic thalamus at the junction of its posterior third with its middle third, the adjoining caudate nucleus being a little involved. In Bernhardt's case there was a softening in the corpus striatum, destroying the external part of the lenticular nucleus and the neighbouring white substance, and there was a focus of softening at the occipital termination of the lateral ventricle. In one of Féré's¹ cases there was an old hæmorrhagic focus in the hemisphere, external to the lenticular nucleus, cutting the sensory crossway posteriorly and reaching forwards to the 3rd frontal convolution; the two posterior thirds of the internal capsule and the corresponding portion of the lenticular nucleus were of a brownish-yellow colour. Fürstner² reports the case of a general paralytic, far advanced in dementia, who presented symptoms of right hemiparesis, right hemianæsthesia, and advanced right amblyopia; there was a small focus of softening in the posterior part of the left internal capsule, as well as a slight pachymeningitic hæmorrhage, and atrophy of the left frontal lobe.

Amblyopia presenting the same characters as that just considered also occurs in cases of lesion of the cerebral cortex and medulla. Féré³ reports a case in which there was softening of the left angular and supramarginal gyri and of portions of the superior parietal and paracentral gyri; the softening extended inwards almost to the lateral ventricle. Demange⁴

¹ 'Archives de Neurol.,' ix. p. 222.

² 'Archiv f. Psych.,' viii. p. 170.

³ Féré, 'Amblyopie croisée,' p. 205.

⁴ Cited by Gowers, *op. cit.* p. 16, and 'Revue de Méd.,' 1883, p. 391.

gives a case coincident with extensive softening of the cortex and adjacent white matter of the outer surface of the right hemisphere; the supramarginal convolution, angular gyrus, and the whole of the occipital and temporo-sphenoidal convolutions on the outer surface were among the parts involved. We shall only mention one more, a case of Petrina's,¹ in which there was lesion of the cortex of the right occipital convolutions, especially the 2nd and 3rd.

What shall we say then? Can amblyopia be attributed to disease of the posterior portion of the internal capsule and the radiations springing from it? Can it be caused by limited lesions in the cortex? Ferrier and Yeo's² experiments on monkeys would show that lesion of the angular gyrus of one side causes amblyopia of the opposite eye, and we notice that Gowers accepts this conclusion as probably applying to man. The facts, however, do not seem at present to warrant any conclusion beyond the general statement, that unilateral lesions of the occipital lobes, and the posterior portions of the parietal and temporo-sphenoidal lobes, are often associated with double amblyopia, most marked on the side opposite the lesion.

That amblyopia results from lesion of the posterior portion of the internal capsule is regarded by many as a fact so well grounded, as to be beyond the reach of criticism or doubt; buttressed, as it is, by the results of the experimental division of the internal capsule, by the facts of hysterical hemianæsthesia, and by post-mortem investigations in cases of the similar cerebral hemianæsthesia. There is certainly a strong case in favour of this view, but by no means so strong as to leave no room for doubt. Let us glance at some of the difficulties attending its acceptance. In the first place it is strange that in a functional disorder, such as hysterical hemianæsthesia, we should have functional impairment of a medullary tract so limited as the posterior part of one internal capsule. Then we are struck by the paucity of the cases of amblyopia with observed lesion of the internal capsule, which is all the more striking when we consider how frequent amblyopia is in nervous diseases. Thomsen and Oppenheim³ are the latest

¹ Féré, *op. cit.* p. 211.

² 'Philosoph. Trans.,' 1884.

³ 'Archiv f. Psych.,' xv.

inquirers in this field, and a very careful and prolonged investigation has shown them that amblyopia (especially bilateral peripheral contraction of the field of vision) is found in the most various conditions. It is the most constant symptom of the group of sensory and sensorial anæsthesias and hemianæsthesias. It occurs not only in hysteria and hysterо-epilepsy, but in epilepsy, alcoholism, neurasthenia, chorea, and many other organic and functional diseases of the nervous system. But not only is it found in widely different cases, in the same case it shows the greatest variations. It fluctuates from day to day, and increases or diminishes according as the general mental condition departs from or approximates to the normal. Further, the amblyopia is bilateral, and, though sometimes unequal on the two sides, is often equal. In amblyopia, in fact, we have to deal with a symptom that is more often functional than organic, and a symptom that displays remarkable variations from time to time as regards its constituent elements of visual acuity, field of vision for white and field of vision for colours, as well as in respect of the relative degree of impairment of each eye. With so unstable a symptom, the greatest caution is necessary in determining its causal lesion, and for our part we do not think that the connection between lesion of the posterior third of the posterior limb of the internal capsule and amblyopia has as yet been satisfactorily made out. At the same time we cannot but admit that, along with the symptoms of unilateral lesion of a hemisphere, there is frequently double amblyopia, most marked on the side opposite the lesion.

We are then confronted with two series of cases; on the one hand, unilateral cerebral lesions producing hemianopsia, on the other, amblyopia. It will be observed that in each, both eyes are affected; and that in each, the vision of the eye on the side opposite the lesion is more affected than the other. There remains, however, the great difference, that the limitation of the visual field is at one time concentric and at another lateral. How shall we reconcile the two classes? Formerly the knot was cut by the advocates of amblyopia denying the existence of cerebral hemianopsia, and *vice versâ*. Now, however, the existence of each is admitted by all, and various

schemes have been propounded as a solution of the difficult problem. We need only refer to Ferrier's,¹ Féré's,² Grasset's,³ and Sharkey's.⁴ None of these can be regarded as quite satisfactory, and we would suggest the following as affording perhaps a clue to the solution:—Cerebral hemianopsia results from the *destruction* of the visual cortical centre, or the optic radiations that enter it from the lower ganglia. When, however the visual cortical centre or the optic radiations are not destroyed, but only *impaired in function*, amblyopia results; the opposite hemisphere sympathising, owing to its bilaterally associated action, or as a consequence of the lesion of its commissural fibres, and so causing slight amblyopia of the corresponding half-field of vision.

Cases are not very rare in which there is combined hemianopsia and amblyopia, lateral as well as concentric contraction of the visual field. What is the localisation of the lesion in such cases? It has been suggested that it is so situated as to affect at once the posterior portion of the internal capsule and the adjoining optic radiations of Gratiolet, or the corpora geniculata and the central end of the optic tract. Féré⁵ gives a case with lesion at the posterior part of the internal capsule, implicating the optic thalamus slightly, and other cases will be found in Wilbrand's 'Hemianopsie.' The cases are neither sufficiently numerous nor clear to permit of a definite conclusion being drawn.

Amaurosis.—Disease of the optic nerve is admitted by all to be a frequent cause of unilateral amaurosis. But when we advance beyond this, and ask whether unilateral amaurosis can be produced by any more central unilateral lesion, we are at once in a region of doubt and dispute. We are not acquainted with any case which shows the effect of destruction of one tubercle of the anterior corpora quadrigemina on the field of vision. We are not aware of any case of amaurosis from disease of the posterior portion of either half of the optic thalamus, or of the internal capsule. Cases have, however,

¹ 'BRAIN,' Vol. III. p. 467.

² 'Amblyopie croisée,' p. 232.

³ 'Montpellier Méd.,' 1883.

⁴ 'Ophth. Soc. Trans.,' iv. and 'BRAIN,' Vol. VIII. p. 137. ⁵ Op. cit., p. 223.

been described, in which amaurosis of one eye has been attributed to disease of the opposite occipital lobe. Thus, Fürstner¹ reports a case of right amaurosis in a demented general paralytic lasting from August till death in December; no closure of the lid, etc., was obtained on thrusting objects up to the right eye, while prompt reaction occurred with the left eye: there was general atrophy of the left cerebral hemisphere, and the pia mater was adherent to the softened cortex over nearly all its convolutions, including the occipital convolutions; there was also a softening in the left corpus striatum, which extended into the adjacent part of the optic thalamus; the pia on the right side was opaque, but non-adherent; the optic nerves were healthy. In another case, there was a similar condition of left amaurosis, but the reaction to retinal impressions seems to have varied with considerable limits: the chief post-mortem result was adhesion of the pia to the cortex over nearly the whole of the right hemisphere, including the inferior parietal lobule, 1st occipital, and cuneus; on the left side there were no adhesions behind the frontal lobe. Stenger² gives a case of right amaurosis, occurring a few days before death, in a patient already suffering from mental blindness; but the pathological changes were too diffuse to allow of any conclusions as to localisation. Zacher³ reports eight cases of transient unilateral amaurosis, coincident with attacks of unilateral motor paresis, and attended with blunted sensibility and more or less stupor; but he gives no post-mortem results. We cannot on such evidence accept the view, that amaurosis of one eye can be caused by disease of the opposite hemisphere.

Double amaurosis is found in disease of both optic nerves and both optic tracts. A solitary case of Bastian's⁴ would indicate that it may also occur in disease of the anterior corpora quadrigemina; the patient, who suffered considerably from stupor, was blind during the last fourteen days, and at the autopsy a patch of softening was discovered almost limited to the anterior corpora quadrigemina. If this case receives confirmation, we shall have to regard these ganglia as portals

¹ 'Archiv f. Psych.,' ix.

² Ibid. xiii. p. 228.

³ Ibid. xiv. p. 492.

⁴ 'Paralysis from Brain Disease,' p. 115.

through which optic impressions must pass before we are conscious of them. At present it is an open question whether they may not pass direct to the cerebrum, or at all events to the optic thalamus. Double amaurosis has also been observed in disease of both occipital lobes, complicated with various other cortical lesions (see cases by Moore and Chvostek).¹ Fürstner² reports a transient attack of double amaurosis in a case which presented, before and afterwards, marked impairment of vision: there were two almost symmetrical patches of softening in the occipital lobes. Stenger³ gives a case of left hemianopsia, followed by double amaurosis during the last four weeks of life, in which the chief lesion was marked atrophy of the occipital lobes, with firm adhesion of the injected pia to the softened cortex; there was also atrophy of the corresponding medullary tracts, down to and including the posterior portion of the optic thalamus. The same author gives two other cases of double amaurosis, lasting respectively ten and four days, in patients having symptoms of mental blindness, but the post-mortem revealed only general, diffused changes. Shaw⁴ reports a case of blindness and deafness, associated with complete atrophy of the angular gyri and superior temp.-sphenoidal convolutions of both sides; the optic nerves, however, showed increase of connective tissue and atrophy of fibres.

We may mention here a case of Bernhardt's,⁵ of right amaurosis, and concentric contraction of the left field of vision, in a patient suffering from aphasia, right hemiplegia and hemianæsthesia; there was a lesion in the left corpus striatum, affecting chiefly the lenticular nucleus and the adjoining medulla; the optic tracts and nerves were normal. Double amaurosis is by no means infrequent as a transient and functional disorder; it is probably due in some cases to retinal affections, and in others to vascular changes affecting the optic strands or their central terminations; but its pathology is little known. Double amaurosis may result from a combination of two hemianopsias or two amauroses, so that it

¹ Wilbrand's 'Hemianopsie,' p. 155.

² 'Archiv f. Psych.,' viii. 165.

⁴ 'BRAIN,' Vol. V. p. 430.

³ Op. cit.

⁵ Wilbrand's 'Hemianopsie,' p. 126.

cannot be used in argument to favour either the hemianopic or the amblyopic view.

Objects seen and handled, and other reflexes.—In the first part of this paper we enumerated a series of visual reflexes (Nos. 3 to 10) of gradually increasing complexity. Important as we deem some such division clinically, it would be futile at present to attempt to localise each of these reflexes. We shall therefore treat them as a whole, noticing separately only special points. The peculiar symptoms observed when these reflexes are absent or impaired, it being understood that objects can still be fixed and followed, comprise what is termed psychical or mental blindness (*Seelenblindheit*). What is the localisation of the lesion in this disease? The cases reported do not enable us to fix a sufficiently precise localisation; they do, however, point to the cerebral cortex as the seat of the lesion, and by a process of exclusion, more especially to the cortex of the posterior parts of the parietal lobes and the cortex of the occipital lobes. In Reinhard's case¹ there were hundreds of cysts scattered through the cerebral cortex; they were especially abundant in the parietal and frontal lobes, a few only being found in the temporo-sphenoidal, and none in the occipital; a few were found in the corpus striatum and optic thalamus, and in the cortex of the superior cerebellar lobe. In one of Stenger's cases there was adhesion of the membranes over the convex surface of the hemispheres, atrophy of the 3rd left frontal convolution, and dilatation of the lateral ventricle, especially in the posterior cornu: in another, general adhesion of the pia over the convex surface of the hemispheres, and at the base of the brain adhesion over the occipital and temporal convolutions; also recent hæmorrhagic extravasations into the pia over the inferior parietal, inferior frontal, and superior frontal gyri of the right side: in a third and fourth, only diffuse changes, such as atrophy and dilatation of ventricles, the membranes being non-adherent. We may mention here a not very clear case of Chaffard's,²—sudden loss of speech and intellectual faculties, patient understanding

¹ 'Archiv f. Psych.,' ix. p. 147.

² 'Revue de Méd.,' 1881. See Crouigneau's 'Vision Mentale, 1884.'

nothing, right ptosis, eyes did not follow objects which crossed field of vision, and yet were not amaurotic, for patient seized objects within reach when he needed them; death in a few days; a focus of softening in left hemisphere, destroying supramarginal and angular gyri, reaching above to intraparietal fissure, and bounded below by fissure of Sylvius and posterior end of superior and middle temporo-sphenoidal convolutions.

In typical cases of mental blindness, both eyes are affected, and the lesion is bilateral. Zacher, however, points out that in his cases (there was no autopsy) the symptom was always associated with dysphasia and right-sided motor symptoms, so that it is quite possible that the left side of the hemisphere exercises a predominant influence in the causation of the symptoms.

It may be well to glance for a moment at the different views that have been taken of the so-called mental blindness. Munk deems it a loss, not of visual perception, but of visual conception; the objects are seen, but not recognised; the associations they used to arouse are no longer aroused. Goltz thinks the phenomena may be explained by supposing a greatly diminished sensibility to colour; everything appears grey and misty, and hence objects are not distinguished. Mauthner¹ attributes the phenomena to destruction of the cortical area presiding over direct (central) vision, and the consequent uncertainty as to the form of objects. Wilbrand thinks they are due to various combined defects of the visual fields, and to the resulting embarrassment of vision. To us it seems impossible to accept any one of these explanations to the exclusion of the others. Doubtless in some cases impairment of visual acuity and colour-sensation, especially when there are other mental weaknesses, produces symptoms of mental blindness, and we have an amblyopic form of this affection. Sudden disturbances in the fields of vision, by leading to temporary visual confusion, may likewise be expected to produce similar results. On the whole, however, Munk's explanation appears to us the most satisfactory, and the most generally applicable in typical cases of mental blindness.

¹ See Stenger, *op. cit.*

We have seen that there are reasons for connecting mental blindness (*i.e.* loss of reflexes, Nos. 3 to 10) with lesion of the cortex of the posterior parts of the cerebral hemispheres. The question immediately presents itself, Is intactness of the cortex necessary for the exhibition of these reflexes? To evoke them, is it not sufficient that the optic thalamus and corpus striatum are uninjured? For if not, what is the function of these basal ganglia? Let us take one of the lowest and best organised of the reflexes, that of blinking as a result of retinal impressions. Is this always a cortical reflex? Nothnagel¹ cites cases to show that where reflex blinking is retained, with inability to blink voluntarily, the optic thalamus and its radiations into the hemispheres are uninjured. But this still leaves open the question whether the activity of the cortex cerebri is required or not. From the fact that absolute blindness, partial or total, with absence of blinking and similar reflexes, is produced by cortical lesions, we incline to think that the visual area of the cortex is necessary in man for even these simple reflexes,² and much more of course for the higher reflexes, such as those concerned in avoiding obstacles in walking, and in the acquired automatic acts (we have already pointed out that it is still unsettled whether such low reflexes as the retino-ciliary and retino-ocular do not in man require the agency of the cortex). There is a theory that when acts are repeated sufficiently often to become automatic, they are banished from the cerebral hemispheres to the basal ganglia. It is difficult to accept this theory. It seems more probable that the reflexes are still cortical; but we are quite in the dark as to the parts respectively played in these acts, by the visual area of the cortex cerebri, and the optic thalami or other ganglia.

Word-Blindness.—The localisation of the reflex by which objects are seen and named, is probably almost identical with that by which letters and words are seen and read. It will be sufficient, therefore, for us to consider the localisation of the

¹ Op. cit. p. 255.

² Wilbrand's theory of the localisation of the three centres for light, form, and colour-perception in the occipital cortex has been so recently noticed in 'BRAIN,' that we shall merely refer the reader to Dr. Anderson's review in 'BRAIN,' Vol. VIII. p. 264.

lesion that produces word-blindness. The subject is a comparatively new one, and it is not surprising that the observations on which our present judgments are founded are few in number, and not very satisfactory. They agree in this, however, that the lesion was always in the left hemisphere. In Broadbent's case¹ (word-blindness, inability to name objects), there was a lesion in the white substance lying between the posterior end of the fissure of Sylvius and that part of the ventricle from which the descending cornu starts, which isolated more or less completely the angular gyrus; there was another lesion in the superior temporo-sphenoidal convolution. In a case of Magnan's² (right hemiplegia, frequent inability to name objects or to read, and, latterly, loss of power of recognition), there were lesions in both hemispheres, the most important being a large softening occupying the left occipital and temporo-sphenoidal lobes, and extending to the base of the superior and inferior parietal lobules. In Déjérine's case³ (embarrassment of speech, right hemiplegia and hemi-anæsthesia, inability to name simple objects, inability to read or to understand the few words he could read—the symptoms varying considerably from time to time), there was a tumour in the white matter of the left inferior parietal lobule, destroying the temporo-sphenoidal medullary fasciculus as well as that of the inferior parietal lobule; and affecting the cortex of the lower half of the inferior parietal lobule and the posterior part of the superior temporo-sphenoidal. In d'Heilly and Chantemesse's case⁴ (aphasia, inability to name correctly and to read, agraphia, word-deafness), there was softening of the upper half of the left superior temporo-sphenoidal convolution in its posterior half, and of the adjoining angular gyrus and a small portion of the supramarginal convolution. The lesion that produces word-blindness may be provisionally placed in the angular and supramarginal gyri of the left side. Further investigations must elucidate its more exact localisation, and decide whether the lesion may be confined to the cortex, or must affect the underlying medullary tracts.

¹ 'Med-Chir. Transacs.' 1872.

² See Mlle. Skwartzoff's 'Aphasie,' p. 48.

³ Mlle. Skwartzoff's 'Aphasie,' p. 53, and 'Prog. Méd.' 1883.

⁴ Crouigneau's 'Vision mentale,' p. 124, and, 'Prog. Méd.' 1883.

They must also tell us the effects of lesions of the corresponding centre on the right side.

We can cite no cases to illustrate the localisation of the higher visual reflexes. We can only surmise that the changes will be microscopic, and will implicate the nerve cells of the cerebral visual area. But inasmuch as the higher visual reflexes depend on the associated action and re-action of other sense-centres, with the condition of which they are necessarily bound up, we are far from sanguine as to the success of attempts at localisation in this sphere.

A NOTE ON SPASTIC PARAPLEGIA AND THE TREATMENT OF SOME CASES BY REST.

BY H. B. DONKIN, M.B. (OXON.), F.R.C.P.,

*Physician to Westminster Hospital, and to the East London
Hospital for Children.*

THE clinical study of the motor affection known as spastic paraplegia has induced me to lay considerable stress on the importance of absolute rest as a means of relief or, possibly, of cure in some cases. The anatomical name of *lateral sclerosis* given to cases of this kind, whether further knowledge of pathology may justify it or not, has certainly not been without its drawbacks. Through its influence, perhaps, to some extent the ætiology of the disease remains an unwritten chapter; and the question of treatment is regarded as useless. As is well known, the theory of the connection of the salient symptoms of spastic paraplegia—loss of motor power, spasm and rigidity, and increase of the deep reflexes—with a sclerotic change in the lateral columns of the cord, rests mainly on inference, though to some extent on direct observation. The occurrence of such symptoms on one or both sides of the body as a result of a descending lesion from the brain or cord is established by sufficient demonstration. It was a probable step in inference, considering these facts, that when such symptoms occurred symmetrically, say in the legs, without any history or other evidence of previous disease, the cause might be found in a primary and symmetrical affection of the lateral columns. There is some degree of evidence of the existence of such a primary disease, but at present the morbid entity with which we are concerned is mainly a clinical one.

Spastic paraplegia, characterised as above described, is by no means a rare disease, though it has been looked on as a distinct affection only of late, consequently on its supposed or real dependence on a recently discovered anatomical affection

of the cord. It may be quite right in the present state of our knowledge to regard primary lateral sclerosis as a rare, or at least a not demonstrably common disease; I cannot but think, however, that the statement of a modern author, that "spastic paraplegia is an extremely rare affection which has recently been recognised as a distinct disease," will be admitted to be mainly inaccurate; and that it is probably the offspring of an undue confusion between a set of clinical symptoms which, though but lately described with accuracy, has been of common occurrence, and a supposed, though probable, anatomical concomitant, which has as yet been but seldom found. The term "pseudo-lateral sclerosis," too, given by some authors to cases marked by the same symptoms, which terminate in recovery, and are therefore regarded as functional, is another instance of similar confusion. It is, to say the least, an assumption that lateral sclerosis is the primary and exciting cause of all the cases which do not recover. Clinical observation, on the other hand, would suggest, from the close resemblance which exists between the cases which recover and those which do not, that there is some cause other than primary structural disease of the cord which may at first account for both.

Considering the not very rare occurrence of this set of symptoms, and the small basis of fact on which their anatomical explanation rests, it is premature to treat of "spastic paraplegia" and "lateral sclerosis" as synonymous; and it is certainly most unjustifiable and misleading to give the name of "pseudo-lateral sclerosis" to cases which from their very nature seem to exclude the notion of sclerosis altogether.

As an instance of the confusion to which I refer, I may quote some passages in Dr. Althaus' book on Sclerosis of the Spinal Cord. A case is quoted of spastic paralysis presumably due to syphilis, which is said to have been cured by the usual remedies, the patient dying soon after of phthisis. The point insisted on is that the post-mortem examination showed that "*the symptoms of the spinal disease may disappear when the lesion which caused them is still present.*" But, a few pages further on, this author contends for the existence of what he calls "pseudo-sclerosis," on the ground that there may be

clinically "*all the symptoms of lateral sclerosis, with no lesion after death.*" He quotes a case of his own, among others, where a girl, who had been bedridden for three years, showed, on admission to hospital, all the symptoms of primary lateral sclerosis, but left in three months nearly well; and draws the inference that no structural lesion had existed, but that the case was one of "pseudo-lateral sclerosis." Taking these two cases together, and the comments thereon, we have an exceedingly striking example of confused pathological reasoning.

The cases which show the symptoms known by the name of "spastic paraplegia," but which either from recovery or other reasons are not dubbed "sclerotic," are by no means always obviously hysterical. They may occur in patients who show no other so-called hysterical symptoms. Temporary attacks, again, of weakness of the limbs, with ankle-clonus and other increased "reflexes," are recognisable from time to time after great exertion and fatigue, or after unaccustomed exercise, or strained position, such as carrying weights; or sometimes after a ride on horseback.

Dr. Wilks, in his 'Lectures on Diseases of the Nervous System,' lays stress on the passing nature of some cases of spastic paralysis. He considers that more cases are wanted to place the pathology of this affection on a positive basis, and prefers to describe it from a clinical standpoint under a clinical name. He points out, moreover, that many instances which formerly would have been regarded as chronic meningitis are probably now looked upon as lateral sclerosis; and quotes a case which, from certain concomitants, seemed to suggest a chronic meningitis as its most likely interpretation.

In studying, when possible, the early symptoms of spastic paraplegia, even in cases which ultimately become so marked as to be obviously incurable and support most strongly the view of their being connected with lateral sclerosis, we find the closest analogy to the class of cases above alluded to which are temporary and probably functional in nature. In a very well-marked instance known to me, leg-fatigue and very occasional falling were the first noticed symptoms, the patient being exceedingly active and prone to exertion. "The lateral columns are fatigued," was a metaphorical but suggestive

remark made by an observer of this case. Perfect rest was not enjoined; perhaps it scarcely ever is in affections of this kind, doubtful as is their nature at first; and the case went on till it became as complete an example of spastic paraplegia with extreme rigidity as could be quoted.

In the January number of this Journal for 1883, I made some remarks on the possible functional origin of spastic paraplegia in some instances, the remarks being mainly speculative and suggestive, but partly founded on some observed cases. One case especially attracted my attention at that time, of which I gave a short abstract, the full report having been published in the 'British Medical Journal' for Dec. 9, 1882. It occurred in a young acrobat, whose symptoms were of a marked character, and had a gradual onset, the complaint varying for some time notably and directly with the amount of rest which the patient took. This case passed from my notice, and I have been unable to find him since.

In the last two years I have observed several more cases, and have endeavoured to ascertain the mode of onset, and to try the treatment of absolute rest in bed. The observation which has been made by some writers, of the comparatively frequent occurrence of this affection in muscular men, and those who are constantly on their legs, is quite borne out by my own experience and by inquiries made of others. It has been said to be not uncommon in sailors, and I have known two cases in postmen. In most of the cases I have seen the onset has been gradual; though in two, a sudden and unaccountable fall, without any other symptoms, has been the first thing to attract much attention. The generally healthy condition of those who suffer from this disorder renders the treatment by absolute rest most difficult to carry out; and to this difficulty with hospital patients is added that of a necessarily long stay in the wards, and the great improbability of strict rest being maintained when the patient goes to his own home, or to a convalescent institution. I have therefore but scanty evidence to bring forward as to the value of this treatment, but such as it is, it points, I think, to the advisability of its being indefinitely persevered in.

The following short notes of four cases I have had lately in

my wards at Westminster Hospital, give some illustration of one or other of these points: namely, the mode of onset, or the possible value of treatment by rest:—

CASE I.—R. F., æt. 49, a field labourer, admitted March 1884. Has been a strong muscular man and always hard-working. He suffered from weakness in the legs, first noticed some six months before admission; and for about three years before had been somewhat losing flesh. He had lost about one stone and a half in that time. The stiffness of the leg was noticed on getting out of bed in the morning. The classical symptoms were all well marked. The patient remained in hospital only a month, and left unimproved.

CASE II.—W. S., æt. 56, cement worker, admitted April 24, with the symptoms well marked. In addition he had some numbness and an occasional feeling of ‘pins and needles’ in the legs; sometimes giddiness; also frequent micturition. The patient was otherwise very healthy, and had a good previous history. About three or four months before admission he noticed his left leg beginning to “drag” as he walked, and soon afterwards the right as well. He can now walk but little; soon gets tired, and his gait is typically spastic. He went out with only the motor symptoms; but these somewhat improved after complete rest for about three weeks. This patient had been in the habit of walking for from twelve to fourteen hours daily for many years.

CASE III.—J. P., a field labourer, æt. 55, admitted Oct. 13, 1884. In this case there was marked spastic gait and difficulty in walking. In addition the patient was subject to headache and giddiness. Four months before admission he fell to the ground when working, but did not lose his senses; he thought he had a sun-stroke. He had a similar fall about three years before. The weakness of his legs was only noticed by him at the time of the last fall, to which he attributed it. The symptoms were very marked on admission; the general health was very good. He was kept at perfect rest for rather more than three months, and went out very greatly improved; the rigidity being much less, the walking power much better, the knee-jerk far less, and the ankle-clonus not to be elicited.

CASE IV.—A. Me. C., æt. 51, admitted Nov. 14, 1884. This patient was an engineer in a cable-laying steamer. He had had good health, with the exception of ague in China not long before he began to

complain of his present affection eight months ago. He had been a very moderate drinker, and had not had syphilis. His family history was very good, not neurotic. His occupation kept him constantly on his legs, very often from 18-20 hours out of the 24. On admission he looked perfectly well; his legs trembled, and he could not stand without support. On attempting to walk, his feet clung to the ground. The knee- and ankle-joints were markedly rigid, those on the right side being the worst (this was the side on which he first noticed loss of power). The knee-jerk was much exaggerated on both sides; and there was also knee-clonus. The ankle-clonus was extremely well marked, about equally on both sides. The special senses were normal, nor was there any other sign or symptom of disorder, nervous or otherwise. He was put to bed, and kept there almost continuously for four months. At first there was frequent twitching of the legs as he lay in bed, the feet being visibly drawn up sometimes. After about a month he was tried out of bed, and it was found that he could not only stand, but also walk alone a little. He had the faradic and galvanic currents applied to his legs soon after admission twice or thrice for diagnostic purposes, to which he attributed the departure of the twitchings, which soon ceased. On Jan. 14, 1885, a still greater improvement was noticed, though the 'jerks' were still marked. He had occasional pain, sometimes severe, in the course of the right sciatic nerve.

On March 11, the patient was noted as much better, though still complaining of the pain in right leg. He could walk the length of the ward without support, and raised both feet from the ground. There was no tremor when the feet were placed on the ground, and very little stiffness of joints. The jerks were less marked.

On March 24 he was again seen to be improved, all the symptoms being lessened. Soon after this he left the hospital, and had a prolonged sojourn at a convalescent home. On August 6, 1885, he was seen at Westminster Hospital. He had been resting all the time, but not so completely as had been advised. He complained still of occasional pain in his right leg. But his walking was so much improved that it in no way suggested the notion of spastic paraplegia; there was no rigidity of any of his joints, and the knee-jerk was very much less than on discharge. The ankle-clonus could still be evoked, though but slightly, and temporarily.

This is the only case which I can quote as one of spastic paraplegia, well marked, and of the kind which might, as well as any, be called before death lateral sclerosis, which has improved in such a notable degree as to demand attention.

But in at least one more of the above cases, besides others that I have seen, there seems to be a strong hint at the possibility of *complete and prolonged* rest being found to be very valuable in the treatment of early cases of this kind, and perhaps also to throw some little light on the ætiology of the affection in some instances. However this may be, it seems clear at present that we must bear in mind that we have not done with spastic paraplegia by calling it lateral sclerosis, considering the cases which recover, whether they are quite obviously functional or not; and must believe, rather, with Friedreich, Wilks and others, that the symptoms may occur under various forms of disorder or disease of the nervous system.

ON PROFESSOR HAMILTON'S THEORY CONCERNING THE CORPUS CALLOSUM.

(*Preliminary Note.*)

BY C. BEEVOR, M.D., M.R.C.P.,

Assistant Physician to the National Hospital for the Paralysed and Epileptic, and to the Great Northern Hospital.

IN the 'Proceedings of the Royal Society' for February 23rd, 1884, later in the 'Journal of Anatomy and Physiology' for July last, and also in the last number of 'BRAIN,' Professor Hamilton has published his researches on the corpus callosum, which lead him to believe that the current idea of the corpus callosum is entirely erroneous.

Briefly stated, his views may be expressed as follows:—

The corpus callosum is not an interhemispherical commissure at all, but is the decussation of the cortical fibres in their progress downwards to become connected with the basal ganglia. The course of these fibres begins in the cortex of one hemisphere—say the right,—the fibres then pass into the corpus callosum, decussating in the middle line with the corresponding fibres from the left hemisphere, and so cross over to the left extremity of the corpus callosum, and then go upwards, outwards, and turn downwards towards the lenticular nucleus. Here the fibres divide; the most inferior and internal pass into the inner capsule, while the most superior and outer run into the external capsule of the left side; the same arrangement occurs in the frontal and occipital parts. A mere fraction of the fibres passes from the right cortex direct to the posterior limb of the right internal capsule, and forms eventually the well-known pyramidal tract which decussates in the medulla.

The conditions stated above are so different to what has usually been considered to exist, and the results to clinical

medicine would be of such great importance, that I do not think the subject can be approached without systematically working the question out, and for this purpose I have been engaged for the last twelve months in endeavouring to trace the connections of the corpus callosum.

I think the key to the whole question lies in this point:—Is it possible to trace a single fibre from the corpus callosum into the internal capsule?

I propose in this paper to give only a very short notice of my work in this direction, as I hope to publish a full account in the next number of 'BRAIN.'

The preparations used by me have been made from the brains of man, but especially from those of the monkey and the marmoset, which, from its small size, is more easily manipulated. The brains have been hardened in bichromate of potash, followed by methylated alcohol, and have been cut, after imbedding in collodium; I have also employed the paraffin method used at Leipzig. I have used Schanz's microtome. The sections have been stained by Weigert's hæmatoxylin method, and especially the acetate of copper method described by him in the 'Fortschritte der Medicin,' 1885, No. 8.

I have been careful not to use any manipulation which might disturb or alter the position of the fibres, and have relied entirely on the microscopical examination of the thinnest possible sections.

From the examination of my specimens, which are cut in a frontal direction, I cannot find a *single* section in which the fibres from the corpus callosum pass into the internal capsule, and I therefore hold the opinion that the accepted view is correct, viz. that the corpus callosum is an interhemispherical commissure, that the corona radiata does exist as such, and that all the fibres do pass from the cortex direct to the internal capsule.

In the drawing which Professor Hamilton publishes in the 'Journal for Anatomy and Physiology' for last July, he figures the "crossed callosal" fibres, which he thinks come from the outer extremity of the corpus callosum, and pass upwards, outwards, and downwards to the internal capsule; but

his drawings do not demonstrate these fibres actually issuing from, or passing into, the corpus callosum, they are represented as commencing, or ending, abruptly in the centrum ovale away from the corpus callosum.

In my sections these fibres are well shown by the microscope, but with this great difference, viz. that these fibres clearly pass on to, or are continuous with, the convolutions of the marginal region of the same side; they course side by side with, and in part pass through, the fibres turning upwards from the corpus callosum. They do *not* pass into the corpus callosum.

This is especially well shown in the brain of the marmoset. In frontal sections it can be seen that the fibres in the centrum ovale, after they issue from the internal capsule, are not so curved as in man and the higher apes, but they run almost straight upwards and inwards, so that it can be demonstrated in all my sections that these fibres do not present any curve or bend inwards towards the corpus callosum, but can be traced to the upper convolutions of the same side, and therefore it is impossible to consider that these fibres can come from the corpus callosum.

Moreover, in all my sections the fibres of the corpus callosum can be seen radiating through the fibres from the internal capsule, the middle fibres of the corpus callosum being at right angles to, while the upper and lower fibres are oblique to, the direction of the capsular fibres.

With regard to Professor Hamilton's views of the decussation in the middle line of the corpus callosum, I should state that, owing to the fact that a number of fibres have to be gathered from all directions into a transverse commissure, it is easy to see how a decussation may be imagined to exist, but I must maintain that no general decussation can actually be demonstrated.

Lastly, in addition to the evidence furnished by my own microscopical investigations, it appears to me that Professor Hamilton's views as to the structure and connections of the corpus callosum, are in absolute contradiction to some of the best-established clinical and pathological facts, as well as to those of comparative anatomy

ON A NEW INDUCTION APPARATUS.

BY DR. E. TIEGEL (NEW YORK),

Formerly First Assistant to Prof. GOLZ, Strassburg; late Professor of Physiology in the University of Tokio.

ALL observers agree that the various kinds of induction apparatus in use at present have imperfections. The physiological effect of the make, or closure shocks, is not as great as that of the break, or opening shocks; the former are, besides, unequal among themselves. Up to this time it has been an impossibility to make even closures at any one fixed rate of time. Moreover, it has been an exceedingly complicated matter to graduate the strength of the opening shocks, a condition as yet scarcely fulfilled in a manner satisfactory for practical applications.

In the year 1877¹ I made certain observations on the physiological effect produced when we put a condenser into the secondary current of an induction apparatus. I called attention to the observations of Galvani, who during a thunder-storm remarked twitchings of frogs' legs simultaneous with every flash of lightning. By induction the frogs' legs had been charged to a certain degree with electricity, which was lost as soon as the inducing body was discharged. If we want to imitate the experiment artificially, we must connect the frog's legs with the earth, because their surface is too small. My aim was to give to structures, susceptible of electric excitation, a surface sufficiently large to produce an inductive effect. I therefore connected such an irritable structure with a plate of a condenser, the other plate of which was connected with one pole of the secondary coil of an induction apparatus. The other end of the coil and a point of the irritable structure were connected either with each other, or together with the

¹ 'Archiv für die gesammte Physiologie,' Bd. xiv. S. 330.

earth. This arrangement gave a series of very remarkable results, of which I will specially mention the following.

(1.) The closure shocks are physiologically absolutely without effect, whether applied as single shocks, or in a series, as a tetanising current.

(2.) The tetanic curves which result are either high or low, if we connect one or the other end of the secondary coil with the condenser, or, what comes to the same thing, if we change the direction of the current in the primary coil.

(3.) By increasing or diminishing the distance between the two leaves of the condenser, according to a fixed scale, we modify by a uniform graduation of the heights of the tetanic curves.

In Fig. 1, Π means the secondary coil, p_1 and p_2 its two

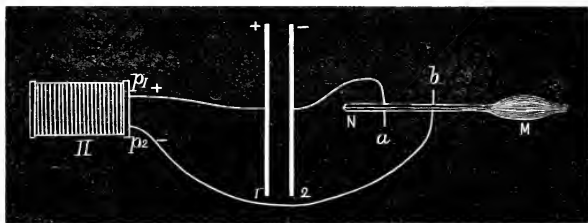


FIG. 1

ends. The plates of the condenser are represented by 1 and 2. Plate 1 is connected with p_1 , and plate 2 with a point a of the structure to be excited. Let us suppose our preparation to be a nerve with the muscle belonging to it. A point b of the nerve is connected with the end p_2 of Π . When we interrupt a current flowing in a due direction through the primary coil, we obtain at p_1 a positive potential of a certain fixed amount, and at p_2 an exactly equal negative one. Positive electricity is conducted to the first plate 1 of the condenser. We have on the other hand negative electricity at p_2 , plate 2, and in the nerve. This negative charge disappears almost instantly and its coming and going is the cause of excitation. Whenever we break the primary current, the same course of events is repeated. By reversing the direction of the current in the primary coil,

the signs of the potentials are changed. We now charge our nerve with positive electricity, whenever we break the current in the primary coil.

If we compare the two kinds of electricity, we observe without exception that, all the other conditions remaining constant, the negative charge produces a more powerful excitation than the positive. At the points *a* and *b* (viz. at the two electrodes) we obtain, besides, a peculiar effect. If we charge the structure negatively, then the electrode with the higher potential has the greater effect, but in charging it, it is positively the electrode with the lower potential which is the stronger. This fact is of very great importance in the practical applications of the apparatus we are about to describe on the living human body. We shall consider this point presently.

My endeavours to make use of these observations for the purpose of constructing a better apparatus for electrical excitation, took a definite shape only about a year ago. At that time I received the aid of Mr. Hermann Lemp, of Berne, at present electrician in Hartford, Conn. Our first aim was to effect the graduation of the strength of stimuli. In my observations on the frog's leg, I was enabled to obtain this result in an easy and pretty manner, by altering the distance between the two plates of the condenser. But in man the surface has to be so great, that only a leaf-condenser is suitable, and this I endeavoured and succeeded in doing. The first apparatus we constructed had for interrupting the inducing current a special electromotor, driven by galvanic cells. The interruption was made by short circuiting. The current could be interrupted either four or twenty times a second. To graduate the stimuli, we used the system resorted to in America by subdividing the secondary coil, using thirty such subdivisions. The apparatus worked very well indeed. It displayed all the peculiarities due to the condenser, besides teaching us two very important facts. On the one hand it displayed, both at the slow, and at the quick, tetanising speed, the phenomenon of the "Lücke" or gap.¹ The influence of the current, both on the motor and on the sensory nerves, increased, when, starting from one layer, we

¹ A. Fick, 'Untersuchungen über elektr. Nervenreizung,' Braunschweig, 1864.
E. Tiegel, 'Pflüger's Archiv für die gesammte Physiologie,' Bd. xiii. p. 272.

successively put into action more and more layers of the secondary coil. But this increase reached a maximum at an early point. From this point, although the galvanometer showed an increase for each new added layer, the effect on the nerves decreased down to a minimum, from whence it soon rose again. It was evident from this fact, that the physiological and the physical effects of the current did not increase and decrease simultaneously. The second important observation was, that the effects of excitation by the secondary current were exceedingly sensitive to alterations in the strength of the primary current—a peculiarity which gave us an excellent means of controlling the physiological effect of the secondary current.

Before describing the methods for arriving at these results, I must point out that it is a serious mistake not to take into consideration alterations of the resistance in the primary coil. Such alterations are always produced if the primary current is made to drive its own interrupting apparatus with ordinary metallic contacts. I shall show later on how, under certain conditions, alterations of $\frac{1}{20}$ ohm may produce a marked change in the exciting effects.

The peculiarities of the new apparatus are the following :—

1. The graduation of the strength of the secondary current is effected by introducing resistances into the primary circuit. Each of these resistances, however, must be so measured, that the amount of the current in the primary coil corresponds exactly to a known and fixed number of milliamperes, which can at once be taken as a suitable measure not only for the physical, but also for the physiological, effect of the electrical stimulus. Experiments which are made on motor and on sensory nerves in the human body have shown, that a gap (“*Lücke*”) never occurs, but that on the contrary the physical and physiological effect of the secondary current grow together. Whether or how far there exists a constant ratio between the two, we leave to be decided by special experiments made for the purpose. In the apparatus, we are describing the greatest amount of current we ever want is 500 milliamperes, or half an ampere ($\frac{1}{2}$ Daniell $\frac{1}{2}$ ohms). As generating elements we employ two gravity cells joined for quantity, or in surface.

For the purpose of being always able to bring the resistance of the inducing circuit to exactly 2 ohms, we send the current through a small sliding rheostat, $r r$, one end of which is connected with the battery, $B B$, and the other with the central of three brass blocks. If we insert a plug in a , the current goes through the primary coil; if at b (leaving a open), the current passes through a Mance's arrangement. In the main circuit we have then the elements $B B$, the rheostat $r r$, a length of wire, $f e$, representing exactly 1.4 ohm resistance, and the galvanometer c . A derived circuit is open through the two wires $c d$ and $d e$, of which each has a resistance of 1.4 ohm. If now the battery and rheostat together have also 1.4 ohm resistance, no current can flow through the bridge $p p$. It

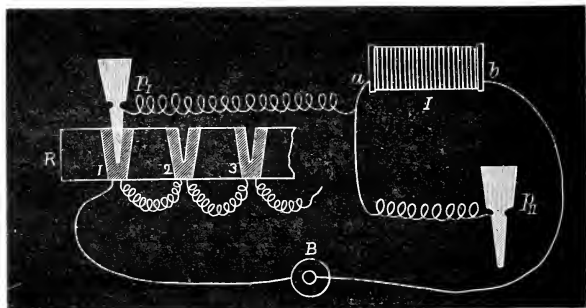


FIG. 2.

will therefore not influence the deviation of the galvanometer c , whether the bridge is open or closed. This point we can always arrive at by shifting the slide s of the rheostat $r r$. Since the resistance in the primary coil is 0.6 ohm, we are always able to bring the strength of the current in the primary circuit to half an ampere.

When this is done, we can graduate the strength of the current in the primary coil by introducing into it known resistances. The successive resistances necessary for a graduation into 100 degrees are thrown in by means of wires, strung between small round brass blocks at equal distances from each other. These 100 blocks are fixed to a large plate of hard

rubber, *r*. In the diagram (Fig. 3), the first three blocks only are shown. They all have cylindrical holes into which fits one of the two plugs, p_1 and p_2 . Both these plugs are connected with the same end of the primary coil by means of a conducting wire. The first brass block, 1, is connected with one pole of the battery, *B B*, the other pole of which is connected with the other end of the coil. When we insert one of the plugs into the first brass block, 1, the resistance in the primary circuit is only 2 ohms, and the current strength, therefore, $\frac{1}{2}$ ampere. But if we insert it into another block, the current will be weaker proportionally to the amount of resistance we have put in. Instead of one plug only we have two, in order to enable us to change the strength of the current,

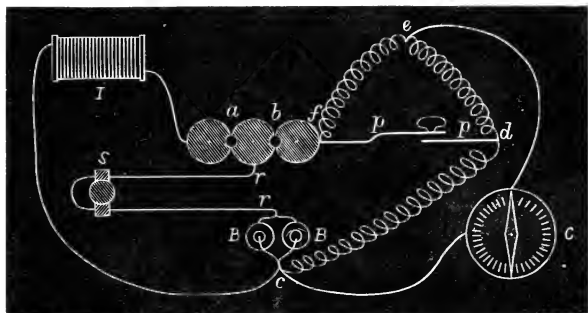


FIG. 3.

without interrupting the latter. The resistances between the single brass blocks have been accurately calculated and measured in the following manner. The apparatus having been finished with the exception of the resistance-board, as much resistance was put into the primary circuit as was necessary to reduce the effect of the secondary current upon the tongue to a minimal sensation. The total resistance necessary to do this was 71.4 ohms, and the strength of the inducing current 14 ma. This amount we have fixed as a minimum for the apparatus. The maximum is, as we know, 500 ma. The difference between the two is 486 ma. For certain reasons we chose the 90th part of this, viz. 5.4 ma.

as the unit for the graduation of the resistance-board. This quantity is called by Mr. Lemp, "the unit of the apparatus." The resistances between the successive brass blocks might have been such that the current strength would have diminished by the amount of this unit from block to block. Experience, however, taught us, that it was better to take into consideration the fact, that in order to obtain a clearly felt increase in the sensation, we must augment the stimulus by a greater ratio as the stimulus itself becomes stronger. Therefore the current-graduation goes first by double, then for single, and last by half units. The brass block which gives the strongest current we have designated as 90. This means that we get from it a current-strength of the

Minimum plus 90 times the unit:

or $14 + 90 \times 5.4 \times 500$ milliamperes.

The following blocks are marked 88, 86, 84, 82, . . . 70. If we put a plug, for example, into the one marked 80, the current strength is calculated in the following manner:

$$14 + 80 \times 5.4 = 446 \text{ ma.}$$

As we have taken an electromotive power of one volt, the total resistance in this case must be 2.25 ohms; in other words, the resistance between blocks 90 and 80 must be 0.25 ohm. Between the subsequent 50 blocks, the strength of the current diminishes every time by a single unit, and they are marked accordingly. Between the last forty blocks the difference amounts only to $\frac{1}{2}$ unit from one another. But all these blocks have been designated after the same system, so that at block 18.5 the strength of the current is

$$14 + 18.5 \times 5.4 = 113.9 \text{ ma.}$$

The last block is marked 0.5, and its corresponding current-strength is therefore 16.7 ma., which again corresponds to a resistance of 59.8 ohm. To get a strength of only 14 ma., we must have a total resistance of 71.4 ohms, or 11.6 ohms, more than the total resistance of the battery, primary coil, and resistance-board added together. These 11.6 ohms are inserted between the last block and the primary coil, so that

we get the minimum current-strength when both plugs are out.

2. The interruption of the current is produced by means of clockwork, which makes the chief axis revolve once during one second. On this axis we have two solid wheels, of which either can be used according to requirement. The smaller of the two is divided into 8, the larger into 40 equal parts. Every alternate part is filled up with hard rubber, while elsewhere the metal of which the wheel is made lies bare. On each of these wheels two metallic contact-pieces are made to rub; the latter are fixed in such a manner that, according to the position of the two appropriate plugs, we simply make and break the current, or instead of breaking, short-circuit it so as to avoid all sparks. The wheels revolve below in oil, so that the contacts are always made under oil.

3. A commutator is provided for reversing the direction of the primary current.

4. The resistance of the secondary coil is 2700 ohms.

5. The surface of the condenser is one meter square.

Instead of attempting a more minute description, we give a perspective drawing of the apparatus (Fig. 4, p. 388).

The following experiments may be made on the healthy human body.

First, it may be shown that the shocks of closure are without physiological effect. For this purpose, place yourself into the secondary circuit, and make and break the primary circuit with the hand. You will never feel any sensation even with the most powerful current, on closing the circuit; on opening, however, the opposite will be the case.

If we allow a current with rapid interruptions to pass through the body from hand to hand, we remark at once that the pain in the skin is very slight in comparison to the excitation of the muscles. Next we find, that after a few seconds on one side the excitation is stronger than on the other. If we now change the direction of the current in the primary coil (and thereby the direction of the opening shocks of the secondary coil), we find again that the amount of excitation is different; it is stronger now at the side where it was weaker before. These differences exist with all current-

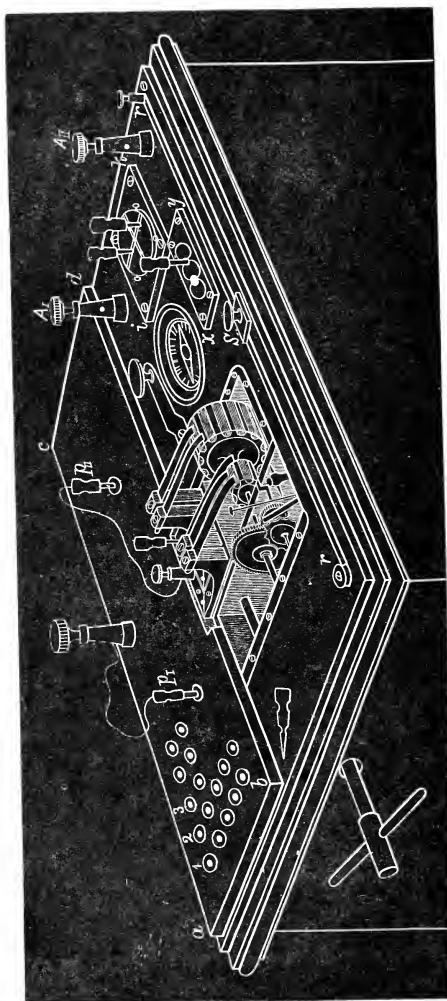


FIG. 4.—It has the shape of a box, and the following parts can be seen on its covering.—*a b c d* is the resistance-board with the holes 1, 2, 3, etc., into which fit the plugs, *p₁* and *p₂*. A few of the holes only are drawn.—The clockwork and interrupting wheels need no further description.—*r* is the rheostat with its slider, *s*—*A₁* and *A₂* are the binding screws for the electrodes.—*k* is a commutator : *x y*, plug and blocks to send the current either through the coil, or through Mance's arrangement.—Elements, coils, condenser, and clockwork are inside the box.

strengths, with slow or rapid interruptions. Waller and Watteville¹ found that if we intercalate increasing resistances into the circuit of an ordinary secondary coil, the effect of the opening currents diminish more rapidly at the positive pole than at the negative. We are entitled to say, that the condenser puts an infinite resistance into the current without thereby interrupting it; the difference in the effect on the two poles thus reaches its maximum by means of our arrangement. This rule is found to hold under the most varied conditions, which, however, have one common point. We must either place both poles upon symmetrical points of the body, and then observe how, according to the direction, the effect predominates at one electrode; or we must put one pole upon an indifferent point of the body, whilst the other rests over a sensitive nerve. In the latter case the effect, whether weak or strong, is localised in the parts to which the nerve is distributed. It is not difficult to find such a current-strength as to become imperceptible. The simplest manner to make the experiment is to take one electrode into the hand, and to fix the other either on the radial or on the ulnar side of the opposite wrist.

It seems that the currents of the new apparatus are especially adapted to excite nerve trunks, while they exert a less influence upon the end organs. If we apply one pole to an indifferent spot on the trunk, and touch successively different points over the head or the limbs, we discover small circumscribed areas and lines where we elicit the liveliest sensation in the district of supply of certain nerves. But if we put the electrode upon the area of distribution itself, the effect disappears. The forehead and occiput, especially, are supplied with so many short nerve branches, that it is difficult to find a place where we do not excite any nerve. Weak currents exert a lasting after-effect when the excitation has been made with the negative potential. The positive potential, on the other hand, produces a lasting soothing effect.

Attentive observers agree that the new current produces a rather different sensation than the ordinary induction currents.

¹ 'Philosophical Transactions of the Royal Society.' 1882.

The sensation is similar to the one produced by the discharge of a Leyden jar.

The condenser has, compared with the body, an infinitely great resistance, so that the resistance of the body need not be considered. We can therefore imagine the whole process in the secondary current to be divided into two parts, a positive and a negative, which parts lie on either side of the condenser. In this sense it may be said that the body is in the part either of positive or of negative potential. The following rule is therefore generally applicable to all observations:—

If the body is in the *negative* potential, the electrode with the *higher* tension will have the *stronger* effect.

If the body is in the *positive* potential, the electrode with the *higher* tension will have the *weaker* effect.

In making a standard experiment let us keep to the same strength of current. We designate with large letters the electrodes of higher, with small letters those of the lower potential, *P* and *N* standing for positive and negative respectively. We have thus: *P*, *p*, *N*, *n*. Simple observations show us that they occupy the following order, from strongest to weakest: *N*, *p*, *n*, *P*.

I have just called attention to the fact, that the resistance in the condenser is infinitely great in comparison with the resistance of the body, and this fact explains another important phenomenon. We place either pole, best of all *N*, on a place where a sensitive nerve lies near the surface, and the corresponding pole is fixed to an indifferent point. Now we diminish the current-strength until we reach a minimal sensation in the area of the distribution. We now note number of units; let us say, for example, that this number is 16. We repeat the observation at another point of the body, where the nerve also lies near the skin, and find again the same number. On myself I have found the same number 16, whether I applied the test on either side of the wrist, or at the elbow or at the eyebrows, &c. In fat people we would expect a larger number. Sixteen units correspond to nearly 100 ma. on my own apparatus.

On increasing the current a little, sensation spreads along

the radial nerve over the whole flexor surface of the forearm. If we now fix the minimal current-strength at several points along this line, as we did it for the wrist, we find that this strength increases as we are approaching the elbow, but differently in different individuals. It is the thickness of the arm, in other words the amount of the derived circuits, which decides the minimal strength sought wherever the nerve does not lie immediately beneath the skin.

The following physical peculiarities of the currents of the new apparatus deserve notice. On account of the condenser the discharge becomes oscillating, and it is therefore possible that interferences in the excitation may occur. In addition to this we have an increase of potential, at the expense of the duration of the current. It is possible that the greater power for nerve-excitation may be due to this cause.

The advantages which the new apparatus presents for the practising physician are the following :—

(1.) We can use it for the purposes of diagnosis. The rate of its interruptions does not produce useless fatigue, and the uniformity of its excitations facilitates the localisation of the motor and sensory points and lines. It also enables us to fix with great exactness the amount of excitability after the method of the minimal stimuli.

(2.) The two potentials have different therapeutical effects. It is found, for instance, that there are certain kinds of neuralgia, upon which the positive potential has a soothing effect, and the negative an irritating one.

(3.) The rate of four excitations a second has been found to be an excellent means to stimulate the vascular nerves, both directly and by a reflex action.

Clinical Cases.

A CASE OF SYPHILITIC DISEASE OF CEREBRAL ARTERIES.

BY EDWARD O. DALY, M.A., M.D. (OXON.), M.R.C.P.

UNTIL the last few years, little mention was made of syphilis affecting the cerebral arteries, either in the literature of syphilis or in the text books of medicine. It is either omitted, or the allusion is very slight. Thrombosis was known to occur not uncommonly during the course of syphilis, and isolated cases had been published, where, together with gummata and thickening of the membranes, a morbid condition of the coats of some of the cerebral vessels had been noticed. It was not, however, till 1874 that the subject was prominently brought forward in a monograph by Heubner.

It is probable, however, that syphilitic disease of the arteries, in its symptoms and results, is as varied and important as that of the membranes, cerebrum or cerebellum; it commonly, as in the present case, leads to thrombosis and, unless the collateral circulation is quickly re-established, to softening results.

J. B., a wood-carver, aged 30, was under notice from October 1883 to July 1884.

His previous history showed he had enjoyed good health till 1876, in the autumn of which year he contracted syphilis. A single sore made its appearance on the prepuce, three weeks after connection, accompanied by enlarged indurated glands, and followed, two months later, by a rash on the chest and arms, and sores on the tongue, mouth and palate. For these symptoms he was treated for a few weeks by a chemist. In June 1878 he married; his wife had had no miscarriages, and gave birth in 1879 to a healthy child, which had remained so since. In the latter part of 1878 he began to complain of attacks of vertigo, lasting at first about a quarter of an hour; their length, however, gradually increased and became constant. With the exception of occasional singing in the ears, no other symptom was complained of till June 1883, when vomiting came on irrespective of food, and worse in the morning and evening; this continued unabated till September. The constant vertigo became gradually

more severe, and at the end of September he was unable to walk without assistance.

On first coming under notice, he presented an anæmic appearance; his features were vacant, but his intellect did not appear weakened. His position in bed was peculiar, and always the same; he sat up with his head and the upper part of his body bent forwards, his chin resting on the sternum. In this position he experienced the smallest amount of vertigo. His walk resembled that of a drunken man. He could not stand with his feet approximated, and dared not attempt to walk without some one at his side. There was no loss of power in the arms or legs; the patellar tendon-reflex was somewhat exaggerated. The pupils reacted to light; the disc appeared normal; there was no squint and no nystagmus.

The hearing on the right side was defective, but the tuning-fork placed on the vertex was most plainly heard in the deaf ear, and was heard longer on the vertex than when placed before the right meatus; it was therefore inferred that the deafness depended on some fault in the conducting media, and not on the nervous apparatus. The heart-sounds were natural, the urine normal; there was no indication of either gastric or hepatic disturbance.

He remained in very much the same condition till November, from which time he gradually improved, and could on January 1st, 1884, walk without help, and staggered only on turning round suddenly. The improvement observed up to January was not maintained, and from the latter end of that month he remained in bed till the day of his death. On the 27th of April he was suddenly attacked with left hemiplegia complete, in the arm, partial in the leg, and some temporary aphasia; there was no loss of sensation, and very slight facial paralysis. From the attack of hemiplegia he seemed to be gradually recovering, when inflammation of the lungs carried him off in a few days, on July 10, 1884.

The most prominent and important symptom in this case was vertigo; it was very constant, increased by the slightest movement, and was complained of even when he was lying down with eyes closed. This is a common symptom in syphilitic disease of the brain, and may be produced, according to Ross, in several ways. Sometimes it depends on syphilitic disease of the auditory nerve, or of the internal or middle ear; sometimes on double vision, dependent on strabismus or general anæmia of the brain, or an irregular supply of blood to it, the result of diseased arteries. Now in this case there was no disease of the internal ear or auditory nerve; double vision was not complained of, and the other conditions which frequently cause vertigo, as cardiac, renal disease,

disorders of the stomach and liver, were absent. It was therefore presumed, as there was a very clear syphilitic history, that vertigo depended on disease of the vessels, and this opinion was strengthened by the sudden appearance of hemiplegia.

It may be mentioned here that headache, which is almost constantly complained of in syphilitic disease of the brain, was absent from first to last. When the patient first came under notice, he presented symptoms frequently prominent in Menière's disease and tumour of the cerebellum.

Vertigo, deafness, and tinnitus are three symptoms invariably present in Menière's disease, which is generally believed to depend on a morbid condition of the labyrinth. The noises in the ear did not, however, recur after his first coming under notice; the deafness was found to depend on some fault in the conducting media, and not on the nervous apparatus. The patient, when he walked, did incline to one side more than another, and objects did not appear to move in one direction more than another, symptoms observed commonly in cases where the labyrinth is affected on one side only. The super-vention, however, of hemiplegia put Menière's disease out of the question.

The reeling gait, the vertigo, the constant vomiting for three months, were symptoms which suggested cerebellar tumour. There was, however, no optic neuritis or any defect of sight; no pain at the back of the head or elsewhere; movements of the eyeballs, refraction of the head and rigidity of the muscles of the back of the neck, symptoms not uncommon noticed in tumour of the cerebellum, were also absent.

The *post-mortem examination* revealed only slight evidence of syphilis in the internal organs. The liver was not enlarged, but its capsule was thickened, and some small and puckered cicatrices were seen in the neighbourhood of the suspensory ligament.

There were also a few scars on the surface of each kidney; the bases of both lungs were in a state of red hepatisation. The heart was normal; there were no atheromatous patches on the lining membrane of the aorta.

When the cranium was removed, the membranes were found healthy. There were no adhesions. The brain presented scattered patches of softening, and on the right side part of the anterior division of the internal capsule had disappeared, owing to thrombosis in the vessels of the lenticular nucleus.

No tumour was found in the cerebrum; but on dividing the cerebellum by a vertical incision into two equal parts, a tumour was observed, occupying its under-surface, which on

microscopical examination was found to be a partially organized blood-clot ; this was of a reddish-brown colour and was of about the size of a large chestnut. The branches of the middle cerebral and basilar arteries were thickened and nodular.

On making sections of the basilar and middle cerebral arteries, the changes described by Heubner were most typically shown. No changes appeared to have occurred in the adventitia ; the muscular coat was in some places infiltrated by a cellular growth, but the chief change had taken place in the intima.

The membrana fenestrata formed a perfectly distinctly landmark on the one side and the endothelium on the other. Between these two there was a large accumulation of spindle-shaped cells, and it was this growth that chiefly caused the thickening.

The lumen of the vessel was narrowed, but free from thrombus. In some places the inner coat was found to be enormously thickened, measuring almost twice as much as the outer and middle coats together.

Although the new formation in this case was almost entirely confined to the internal coat, in other recorded cases the outer coats have been found considerably affected, and this seems especially likely to be the case when the artery is in the neighbourhood of a gumma, or syphilitic inflammation.

In a case related by Dr. Sharkey at the Pathological Society in 1883, the external coat was the chief seat of the disease ; in this case the dura mater was also thickened.

The post-mortem examination showed the cerebral symptoms in the present case depended entirely on syphilitic disease of the arteries and its sequelæ, thrombosis and hæmorrhage, the latter being probably the result of rupture of one of the cerebellar arteries, which doubtless were in a more advanced morbid condition than the arteries examined.

The hæmorrhage into the cerebellum, and the irregular supply of blood to the brain in consequence of the condition of the arteries, fully explained the intensity of the vertigo. Either condition alone causes this symptom ; the presence of both in the same subject necessarily made it more marked.

The limitation of the softening to the anterior division of the internal capsule produced, as was to be expected, motor paralysis only ; there was no disturbance of sensation associated with it.

With regard to the diagnosis of syphilitic disease of the arteries, very few cases, it appears, have been published where the arteries alone were affected. In the great majority of cases, where syphilitic deposit has been observed in the cerebral arteries, inflammatory changes in the membrane or

gummata of the brain-substance have been also noticed. It is probable no symptoms are produced until it causes either local anæmia or softening, the symptoms of which resemble those due to other causes; they will therefore depend on the part fed by the artery which is obstructed, and as thrombosis may occur anywhere, the symptoms may be more varied. Vertigo, noises in the ear, dulness of hearing, *muscæ volitantes*, headache and delirium may precede the paralysis, which will probably occur sooner or later in the case. The paralysis is likely to be sudden, and may pass off rapidly, provided the collateral circulation is quickly re-established, and recur.

Recurring attacks of paralysis when successive paroxysms differ in their character and size is very suggestive of cerebral syphilis (Buzzard), and is especially likely to occur when the arteries are affected.

The patient on coming under notice was immediately put on anti-syphilitic treatment, small doses of mercury and iodide of potassium being given for a few days, the dose of the iodide being quickly increased to thirty grains three times a day. Some improvement followed the administration of the large doses of the iodide, but this was not maintained for more than a few weeks.

The chief points of interest in the case were:—

(1.) The advanced syphilitic disease of the arteries without the presence of syphilitic changes in the brain substance or membranes.

(2.) The length of time over which the cerebral symptoms extended (1878 to 1884).

(It is reasonable to suppose the arteries began to be affected in 1878, when the vertigo was first complained of.)

(3.) The intensity of the vertigo, which caused the patient to be absolutely helpless.

(4.) The small effect of mercury or iodide of potash on arterial disease of such long standing.

A CASE OF HYSTERO-EPILEPSY IN THE MALE.

BY JAMES OLIVER, M.B. (EDIN.), M.R.C.P. (LOND.),

Medical Registrar to the National Hospital for Paralysed and Epileptic.

HYSTERO-EPILEPSY in the well-marked form is, comparatively speaking, rarely seen in this country, either in the male or female, but more especially in the male; in either sex, however, the symptoms may be identical. It is possible, nay likely, that as our knowledge of this disease extends, we shall admit into this category cases which even in the male we previously in our ignorance passed unheeded, or classed far otherwise. Hitherto the phenomena of hysteria have been usually associated with the female habit of body, and we have spurned the notion that the active, well-developed labourer could, whilst in apparently good health, become the victim of this affection. Hysteria is a word which as at present employed is quite unintelligible. It is employed too indiscriminately, and more or less as a "cloak." In the male the symptoms are, as a rule, most intractable; whilst in the female they are vagrant and indefinite facts which often aid us in arriving at a conclusion as to the true nature of the ailment.

Let us ever remember that symptoms apparently hysteroid in character may depend on some organic lesion. We ought therefore to guard against exposing patients unduly to those therapeutic measures so often adopted and found beneficial in the treatment of functional disorders.

CASE.—P. T., aged 36, a diamond cutter, presented himself at the National Hospital, Queen Square, on the morning of June 20th. He appeared to be suffering from left hemiplegia; but whilst talking with me, he had several peculiar spasmodic attacks, affecting the left side, to be hereafter described. I admitted him into hospital, under the care of Dr. Ferrier.

Six weeks previously, patient whilst crossing a street got "jammed between two vehicles." He was much frightened, but uninjured, and feeling no special inconvenience, walked on as though nothing had happened. He had not, however, walked far, when, without warning, he fell unconscious on the ground. He was insensible for about five minutes, but lay perfectly still, there being no convulsive seizure. On regaining consciousness, patient was unable to walk, having lost power over

the left leg ; in fact, there was hemiplegia complete of the left side. For four weeks no change was remarked in the condition. Faradic electricity was then applied to the left arm, and thereupon patient had what he calls an attack of "cramp" affecting the whole of the left side ; with loss of consciousness.

Patient had always enjoyed good health till five years ago, since which time he had become somewhat timid. This he attributes to his having been detained in a burning building, and having had to drop from one of the windows, a distance of several feet from the ground. His sleep, however, during the last five years had never been specially disturbed, nor had his health been impaired, as is usual in the subjects of hysterio-epilepsy after such accidents.

The family history is unimportant, there being no neurosis.

Patient is well-nourished ; there is no wasting. He says he is unable to perform any movement with either the left arm or left leg. The left face seems somewhat retracted, the furrows of this side being altogether deeper than those of the right. When, however, all the muscles of the face are called more or less into action, as on exposing the teeth, the right face appears the more active, the left angle of the mouth hanging lower than the right. He complains of numbness all down the left side of the body, and tactile and painful sensibility here, compared with the right, is markedly impaired. The superficial reflexes, skin plantar, cremasteric, umbilical, and epigastric, are all well marked. There is no ankle-clonus, nor has there ever been even when sought for immediately after any of his convulsive attacks. There is no special increase of the knee-jerk ; the right, however, is apparently greater than the left. There is slight increase in the left wrist tap. The tongue, when protruded, deviates a little to the left. He is unable to detect the difference between salt and sugar when placed on the left half of the tongue, whilst the sensibility of the right is maintained. There is no olfactory anæsthesia, the sense of smell being apparently normal. Patient does not appreciate the tick of the watch with the left ear, neither is the sound conducted and heard in this ear when the watch is applied to the cranium. Both pupils are fairly well dilated ; the left, however, is a little larger than the right. They react to light and accommodation : when exposed to light, they readily contract, but immediately thereafter dilate again. There is total anæsthesia of the nasal half of the left retina, loss of vision in the left temporal field. There is normal vision with the right eye. Patient occasionally wore a pair of faintly blue-tinted spectacles, and then he often remarked that objects appeared green to the left eye.

The attacks of "cramp" affecting the left side are either

spontaneous or produced; in whichever way they arise, however, the course and character are always alike. The eyes assume a peculiar stare, and slowly they, with the head, deviate to the left side, the chin resting on the left shoulder. The thenar and hypothenar eminences of the left hand now become approximated, and almost simultaneously with this movement the middle, ring, and little fingers of this same hand are bent in on the palm. He now becomes unconscious. Up to this time he is conscious of all that is going on, and if engaged in conversation, will continue talking. The hand is then slowly but completely pronated, and the arm at the elbow hyper-extended. Sometimes a twitching of the left index-finger will occur. The left leg is always rigid, but straight. The toes and foot are usually somewhat dorsally flexed, because of an over-action of the muscles governing this movement.

Sometimes in the attacks the whole body would be more or less markedly affected, patient assuming the position of an arc of a circle, but never, as in true tetanus, did the head and heels form the sole points of rest, other parts of the body always touching the bed. Sometimes the right side of the body would be the seat of contortion, either by itself or in association with the left; when the right alone was affected, the attack had invariably been produced. The attitude of the right differed essentially from that of the left limbs, and here the movements were never so intense as on the left side of the body. When the attack affected the right side alone, whether spontaneous or produced, the first thing to be noted was a twitching of the right index-finger, followed immediately by an approximation of the thenar and hypothenar eminences: but no flexion of the fingers, as in the case of the left, the whole hand assuming rather the shape of a cone. Patient now would apparently become unconscious, the hand at the same time being supinated, not pronated like the left. The right leg was but rarely affected; when however it was, its state, like that of the left, was straight and rigid. In the attacks patient occasionally bit his tongue, but never urinated.

Immediately before his attacks patient usually complained of "swimming" in the head, feeling as though he should fall to the left; things about him moving to the left also.

The pulse during a seizure invariably numbered about 78 per minute, but fell immediately the attack ceased to 48; and continuing thus for some time gradually increased in frequency, till it reached its usual number—varying from 70 to 78.

The attacks were either spontaneous or produced. There were four distinct hysterogenic zones, that is to say, points

which when pressed upon did in some way or other exert an influence in the production of the "spasms." These spots were situated over the left brachial, left carotid, left femoral and right femoral vessels. It was not, however, necessary to compress in any way these vessels to produce an attack, as simple pinching of the skin at these spots produced the same result. Here there was no apparent increase in the cutaneous sensibility either, and pressure on places markedly more sensitive developed no attack. Compressing the testicle of either side likewise proved worthless.

The cutaneous sensibility in this, as in the majority of cases of hystero-epilepsy, was very variable; but, in testing, I often remarked that immediately I approached any of the above hysterogene points, patient therewith evinced the incipient signs of an attack, which, however, did not become fully developed, if forthwith the excitation were discontinued.

On one occasion, after two spontaneous attacks which followed so closely upon each other as to be hardly separable, and in which both sides of the body were affected, patient remained speechless for two and a half hours, and appeared to experience at the same time difficulty in breathing and swallowing; the difficulty being more marked with fluids than solids. For many hours after this attack he complained much of a feeling of constriction of the left chest, which did not extend beyond the mid-line.

By applying friction to the forearm during the stage of contracture, but before patient became unconscious, I often succeeded in cutting short an attack.

There was great tenderness in the region of the supra- and infra-orbital nerves, especially the left, yet pressure on these never produced an attack. When the whole body was more or less completely affected by the seizure, the diaphragm invariably participated in the clonic spasm, and the contraction of this muscle would as a rule continue for long after complete relaxation of all the other parts of the body. The diaphragmatic contractions were never accompanied by noise, nor did they ever produce vomiting. If whilst the movements were still continuing in the diaphragm a left-sided attack were induced, the muscle would maintain a clonic spasm during the time the left side of the body was affected, and would even continue for a few minutes afterwards.

Circulatory troubles are frequent in cases of hystero-epilepsy. Our patient often complained of cardiac palpitation, which at times would be so intense as to produce throbbing in all the vessels of the body, but felt especially in those of the head and neck.

TWO FATAL CASES OF TETANUS.

BY W. B. HADDEN, M.D.

CASE I.

H. F., aged 51, a gardener, was admitted into St. Thomas's Hospital, under the care of Mr. Croft, on February 26th, 1885.

For the three weeks preceding admission he had been drinking heavily. He stated that since his youth he had suffered from attacks of bleeding piles, occurring at intervals of about a month. Six days before admission he was thought to have a prolapse of the rectum. It was reduced by a doctor after it had been down three days, and bled rather freely afterwards.

When seen by the house-surgeon, on admission, the risus sardonius was marked, the head was thrown well back, the sterno-mastoids tense, the back arched. The limbs were not then affected. The respirations were 36, occasionally interrupted by spasm; the pulse 112, irregular, weak and compressible. He was sweating freely. A rectal examination revealed some internal piles, and chronic thickening of the mucous membrane. Two hours after admission, chloroform inhalation was tried with marked benefit. After a few whiffs the respirations became regular, the pulse full and steady, and the jaw more relaxed. He usually lay on his face, but after the inhalation he allowed himself to be turned on his back. The chloroform was administered off and on for three hours. He then fell into a quiet sleep, during which the muscles were quite relaxed. But the respite was short. In half an hour he awoke, the opisthotonos returned with violence, and hiccough came on. Chloroform was again given. The hiccough disappeared, and the back became less arched. At 9 P.M. (seven hours after admission) the flexors of the left forearm and the fingers began to twitch. It was noted, also, that before a spasm he raised himself on his hands and knees. At 11 P.M. the pain was very intense, and the attacks began to occur every ten minutes. The paroxysms continued to recur, with short intermissions. Sometimes they came on apparently spontaneously; at other times they were induced by external impressions. The sound of a falling saucer, the chiming of Big Ben, the passage of an enema tube, the

removal of the sheet on which he lay, and sometimes the act of micturition, were sufficient to bring about a paroxysm. During the attacks the respirations became quick and shallow, and the pulse rapid and thready. Sometimes the pupils became dilated, and once priapism was noticed. During the seizures he often passed flatus, and two or three times feces. He perspired much. Between the attacks he dozed occasionally. His chief complaint was of pain at the back of the head and in the abdomen. As the attacks became more frequent and severe, the chloroform was less efficacious, and a larger quantity was required in order to produce relaxation. There was much difficulty in feeding him. Condensed liquid nutriment was given by mouth and by enemata; but he was often disinclined to be fed. Two or three times liquid food was given by the œsophageal tube; but it was not retained. Towards the end the paroxysms became so violent, that now and again he nearly rolled off the bed.

Death occurred at 4.55 A.M., on February 28th (about thirty-eight hours after admission), from a spasmodic attack, affecting the larynx and muscles of respiration. The temperature varied from 99°·4 to 103°.

Post-mortem Examination.—Body fairly nourished. Rigor mortis well marked. Jaws firmly closed. Some old pleuritic adhesions on both sides. Pericardium and peritoneum healthy. The cavities of the heart contained a little fluid blood; the right side was dilated and hypertrophied. The air-passages contained blood-stained mucus. The lungs were bulky, emphysematous at the edges, and there was much blood in the dependent parts. The kidneys and spleen were healthy. The cervical and abdominal glands were swollen. The vessels on the surface of the brain were injected. There was marked congestion on the floor of the fourth ventricle and of the grey matter of the spinal cord, especially of the posterior horns.

The cervical and dorsal sympathetic, the phrenic, pneumogastric and fifth nerves were examined, but no change detected. There were slight external piles, but no prolapse of rectum. Just within the anus the mucous membrane was somewhat swollen, but there was no bruising.

Microscopical Examination.—There was marked dilatation of the vessels of the grey matter in all the regions of the spinal cord. The white matter was comparatively free. In some places the distended vessels had given way and hæmorrhages were seen. In the lumbar region, which was the most affected, they were present in both anterior and posterior cornua, but were more abundant in the former. There was rather a large hæmorrhage into the grey matter midway between the anterior and posterior horns in the dorsal region. The

cervical part was congested, but no hæmorrhage was found. The multipolar cells everywhere were healthy. There was no exudation. The phrenic nerve and the trunk and ganglia of the vagus were normal. The ganglia and cord of the cervical sympathetic were quite healthy, as well as the first dorsal ganglia; but in the dorsal cord the vessels were much distended, and there were numerous hæmorrhages.

CASE II.—C. B., aged 21, a gardener, was admitted into St. Thomas's Hospital, under the care of Mr. Sydney Jones, on April 11th, 1885. Fourteen days before admission he cut the tip of his left forefinger with a wood-chopper. The wound was dressed by a doctor at once. The patient seemed to be progressing favourably until five days before admission—that is, nine days after the injury. He then noticed some sore throat and stiffness about the neck. On the evening of the same day he had some difficulty in opening the mouth, and occasional twitching pains of the back. On admission, the face was flushed and anxious. The tetanic spasms came on every two or three minutes. In the attacks the body assumed the usual arched position, and the risus sardonicus was present. In the intervals he could open the mouth fairly well, and was free from pain or discomfort. His mind was unaffected. The temperature was $99^{\circ}4$. There was an unhealthy-looking wound at the tip of the left forefinger, but there was no swelling of the parts around, and no pain in the course of the nerves. On April 17th, the two terminal phalanges were amputated. He was ordered hydrarg. subchlor. gr. vi., and a mixture of chloral hyd. gr. xx., pot. bromid. gr. xxv., every three hours. The next day it was noticed that he was sweating, that the face was more set, and that the spasmodic action of the muscles of the back was accompanied by great pain. The attacks occurred about four times in the minute. The pulse was 112, compressible. The bowels acted after the calomel. He was ordered ext. physostig. gr. $\frac{1}{8}$ every three hours. It is unnecessary to give the details of the case. The spasmodic fits continued to occur at frequent intervals; the jaw became so fixed that it could only be opened sufficiently wide to allow the tip to protrude; the pulse was quick and compressible, the respirations rapid and shallow. On April 21st an ice-bag was applied along the spine, and morphia injections were used. On April 23rd the physostigma pill was given every hour. After the first day the temperature was always above the normal. In the evening it was usually 1° or 2° higher than in the morning. The highest temperature was $104^{\circ}2$. He died on April 26th.

Post-mortem Examination.—Body fairly nourished. Some lividity about arms. The two terminal phalanges of the left

index-finger have been removed, the distal end of the first phalanx being left bare. The spinal dura mater was healthy; the pia mater injected, especially posteriorly and in the lumbar region. The grey and white matter appeared quite normal, and there was no unusual vascularity. There was no change in the medulla oblongata and floor of the fourth ventricle. The venous sinuses within the skull contained fluid blood. The brain was healthy, except that the puncta vasculosa were well-marked. The blood everywhere was fluid. The organs generally were congested, but exhibited no other abnormal change.

Microscopical Examination.—There was no sign of neuritis of either the plantar or dorsal branch of the digital nerve going to the left index-finger. Sections from the cervical, dorsal, and lumbar regions of the cord were examined and found healthy. The multipolar cells had their normal appearance; there was no congestion and no exudation. The medulla oblongata was healthy.

Remarks.—Case I. shows that in tetanus congestion and hæmorrhage may exist not only in the spinal cord, but elsewhere, *e.g.* in the dorsal sympathetic. Case II. shows that there may be no congestion of the spinal cord or medulla oblongata. Congestion and hæmorrhages occurring in tetanus are, in all probability, accidental, dependent on death from asphyxia. In both cases the multipolar cells were healthy, and there was no exudation, such as is described by some writers. Case II. has an additional negative value; the nerves of the injured finger presented no sign of congestion or inflammation. They were quite healthy. So far, then, as the actual cause of tetanus is concerned, these cases are negative.

CASE OF ALMOST COMPLETE DESTRUCTION OF THE RIGHT HEMISPHERE OF THE CEREBELLUM, WITHOUT DISTINCT SYMPTOMS OF CEREBELLAR DISEASE.

BY GEORGE OGILVIE, B.SC., M.B.,

*Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; and
Lecturer on Experimental Physics at Westminster Hospital.*

Miss W., a young lady aged twenty-two, consulted me on March 4th, 1885, on account of blindness, deafness in the right ear, giddiness, and occasional attacks of vomiting, which were always preceded by severe pain in the back of the head.

The following facts relating to the family history were communicated to me by the patient and her sister:—The father is sixty years of age, and enjoys good health. The mother died at the age of forty-four of some abdominal disease. The family consisted of three boys and five girls. One girl died at five years of age of tubercular meningitis. One son died at twenty-eight of consumption. He had been ill for some years, and a few months before his death he had ulceration of the vocal cords, and became paralysed on the right side. Another boy, who is still living, suffers from epileptic fits. He had convulsions during infancy, attributed to teething, and no fits were again observed until the age of fifteen. Now he has occasional severe epileptic seizures and frequent attacks of the *petit mal*. The other members of the family enjoy good health.

The patient informed me that she had the usual ailments of childhood, and that her present illness began about three or four years ago with symptoms of indigestion. For two or three months at a time she was free from any such symptoms, and then for several days she would experience slight nausea after food, accompanied by variable degrees of pain. These symptoms recurred with greater frequency, and about twelve months ago she began to complain of giddiness, and noises in the head like rushing water, and occasional severe attacks of vomiting, always preceded by a dull heavy pain in the back of the head. After one of these seizures of severe headache, she lay in a more or less torpid condition, and was difficult to rouse. Her sister also informed me that on these occasions her breathing became gradually feebler, until

it was almost impossible to be certain whether she was breathing or not. The pulse also became weaker, and could hardly be felt. It was observed that as the pain in the occiput increased she moved her head slowly backwards and forwards, and finally it seemed to be drawn backwards. She remained in this state about half an hour, when, after copious perspiration, the breathing became apparent, and she gradually regained consciousness. These attacks were latterly more frequent, and towards the end they occurred every other day. Ten months ago her sight began to fail, and for nearly three months she was quite blind. The first symptom she noticed of defective sight was that of double vision; but this disappeared as the sight became weaker. About this time she observed that she was becoming deaf in the right ear, and when I saw her she said she was unable to hear at all with that ear. She never experienced any difficulty in hearing with the left ear. She also stated that when she was giddy, on attempting to walk, she occasionally staggered to the right, but never to the left side. Smell and taste were perverted in such a manner that the smell of the hyacinth and the taste of roast beef, which had previously given pleasure, became intensely disagreeable.

When I saw her on the 4th of March, her appearance and condition were as follows:—She was a tall, full-grown, well-developed, healthy-looking young woman. She walked into my consulting room, guided by her sister, with the gait of a blind person, without reeling or staggering in any way. Owing to her blindness, her expression was, perhaps, at times vacant, but she answered all my questions in a satisfactory and intelligent manner until the end of the interview, when she became somewhat fatigued. Her tongue gave no indication of gastric irritation, and she said her appetite was good. The temperature was normal, and I was unable to discover any evidence of disease of either the heart or lungs. She had no menstrual troubles. As regards the nervous system, I was able to note the following points:—At times she experienced a sensation of cold in the lower limbs, but sensibility to touch, heat, tickling, and pain was normal. Both pupils were widely dilated, and responded very feebly to light and accommodation. There was neither ptosis nor strabismus. On ophthalmoscopic examination, both discs were observed to be white, with well-defined margins, and the vessels were small and shrunken. At the inner margin of the disc of the left eye there had evidently been a choroiditis with resulting atrophy and absorption of pigment. She was scarcely able to distinguish the flash from the mirror in the left eye, but noticed a flash in that eye when the negative electrode of a galvanic

battery was placed over the temple. The ticking of a watch was not heard, even when placed against the right ear, but it could be heard distinctly at a distance of eight to ten inches from the left ear. When the watch was placed on the forehead, she heard it only in the left ear. The tympanum in both ears was apparently healthy. She had no difficulty in estimating the weights of various objects placed in her hands, or suspended from her limbs. The galvanic current produced a metallic taste in the mouth, and she could easily distinguish the taste of salt from that of sugar, and the smell of ether from that of eau-de-cologne. There was no ankle-clonus, and the knee-jerk was normal. She was able to stand firmly when both feet were placed close together, and no swaying motion or inco-ordination of any kind could be detected while walking. The form of the cranium was normal, and no pain was experienced during examination when firm pressure was made over the occiput. As further examination at this time was inadvisable, it was arranged that I should see her in bed next day, in order to confirm my observations.

She retired to bed in apparently good spirits, although somewhat fatigued by her long railway journey to London. In about an hour she awoke with the severe occipital headache, and the respiration and pulse becoming weaker, she gradually sank and died in a few hours, without regaining consciousness. The history and symptoms pointed to a tubercular growth in the right half of the posterior fossa, involving the vagus, the portio mollis, the medulla, and probably pressing on the cerebellum.

At the autopsy, twelve hours after death, I was assisted by Dr. Hughes Bennett. I had some difficulty in obtaining the consent of the friends, and was only allowed to examine the brain. The dura mater was in places adherent to the skull-cap. The superior longitudinal sinus was distended with blood, and on removing the dura mater, the veins of the pia mater were found to be engorged. The convolutions were flattened, and the sulci shallow. Considerable difficulty was experienced in raising the cerebellum, owing to adhesions in the right half of the posterior fossa. As the adhesions were extensive, the greater part of the medulla was unfortunately destroyed. During the removal of the brain a large quantity of sanguineous fluid escaped; and afterwards, on examination, both lateral ventricles were found dilated. The right hemisphere of the cerebellum had been almost completely destroyed by the pressure of a cartilaginous-like tumour, about the size of a hen's egg, which had grown from the dura mater lining the right half of the posterior fossa. Arising from the outer margin of the fossa, near the junction of the

lateral sinus with the petrosal sinuses, it had pressed on the occipital surface of the cerebellum, causing its absorption. It appeared to have passed upwards, inwards, and forwards, so as to destroy nearly the whole of its internal structure. In fact, there was merely a thin layer of nerve tissue covering the tumour. The amygdaloid lobe, the flocculus, and part of the biventral lobe were, however, intact. The vermiform, or central lobe, and the left hemisphere were untouched. The greater part of the medulla having been destroyed during the removal, the relation of the various nerves to the tumour could not be satisfactorily ascertained. The histological examination of the tumour was kindly undertaken by Dr. Hebb, Pathologist to Westminster Hospital, and he pronounced it to be a tubercular mass containing remarkably large giant cells.

Remarks.—The chief point of interest in this case is the destruction of a large amount of one hemisphere of the cerebellum, without the production of *direct* cerebellar symptoms, such as disorders of equilibrium, and co-ordination. This may be explained by supposing that the growth of the tumour was so slow that the other half of the cerebellum was enabled gradually to acquire the power of performing the functions of the whole organ.

The *indirect* symptoms, which pointed to cerebellar disease, were double optic neuritis, vomiting, pain in the back of the head, and occasional staggering to the right side.

It is of interest to note the evidences of pressure on the vagus, in the peculiarities of the respiration and the pulse.

The early symptoms of the disease were such as to lead one to attribute them to hysteria, rather than to serious organic disease, but the knowledge of the tubercular family history was of value in forming a diagnosis.

NOTES AND REMARKS UPON A CASE OF VILLOUS TUMOUR IN THE FOURTH VENTRICLE.

BY J. HARRINGTON DOUTY,

Senior-Assistant Medical Officer, Worcester County Asylum.

THE following case seems worthy of record, both on account of the clinical points of interest which it presents, and also as an example of rare pathology. With the permission therefore, of the Medical Superintendent, Dr. E. Marriott Cooke, I venture to publish the following summary of the notes on the case, and to append a few remarks.

W. S., a lad aged 17; admitted July 12th, 1884. For several months, probably at least for six, before admission there had been some failure of sight and blunting of the mental faculties. During the last few weeks a reeling gait had been noticed.

Notes on Admission.—He is a thin, pale boy, of a tubercular aspect. His lungs and heart act normally. Urine contains $\frac{1}{2}$ albumin, no blood, casts, epithelium or sugar. No apparent palsy of legs or arms. He can grasp, pick up fine objects, and, whilst recumbent, can perform voluntary co-ordinated movements with the legs. When, however, he gets up to walk, he reels (usually towards the right), hurries forward in an uncertain way, and soon stumbles and falls to the ground, unless he reaches some object to which he may cling for support. Says he feels “giddy,” and has a pain at the back of his head. His reeling gait is the same, whether his eyes are open or shut. The reflexes, superficial and deep, are normal. His right arm and hand, and occasionally also the left, are subject to attacks of tremor, which, however, he can control to perform a voluntary act. There is no palsy of facial, lingual, or pharyngeal muscles. He sees very badly, looking always to his own left of the object to get a view of it. If one covers his right eye, the left pupil dilates to the full and is insensible; on uncovering the right eye, the left pupil immediately contracts to about the normal size. Right pupil responds, though slowly and imperfectly, to variations of light and accommodation. The left eye is quite blind. With the right he can see an object if it is held on the outer, or lower side, of the eye. Ophthalmoscopic examination reveals

extensive neuro-retinitis; the left disc is very white and atrophied; the right less so; many white patches are scattered over the fundi, least numerous on the inner and upper parts of the right. The boy hears badly, especially on the left side. The external meatus and the membrana tympani appear normal. Senses of smell and taste unaffected. Ordinary sensation of touch, heat, cold, and electric stimulation are normal. His head is bulged in the temporo-parietal regions. Temperature normal morning and night; pulse 66; respirations 16.

Mental Condition.—All that need be said to describe the state of his mind is that he is in a state of hebetude; he is dull, heavy, and sleepy; rarely speaks, unless roused by shaking, when he can give a better account of his symptoms than one would expect. He says usually, "I am much better," and is of opinion that if discharged from the Asylum to-morrow, he could at once work and earn a livelihood.

These notes give a fair idea of the boy's state when he came here. I may say that the diagnosis arrived at with regard to the cause of his symptoms, was that he had some lesion of, or affecting, the middle lobe of the cerebellum. The exclusion of locomotor ataxy was easy; the absence of pains in the back or legs; the fact that he could perform co-ordinated movements when lying down; the presence of the deep reflexes; the fact that sensation in the legs was neither delayed nor perverted; and the very early sight failure;—were, though not of necessity individually, certainly when taken together, strongly negative to a primary cord lesion. Of cerebellar lesion, on the other hand, there was evidence both *direct*—reeling gait without disorders of sensation, pain, or true "ataxy," and *indirect*—occipital pain, mental hebetude and neuro-retinitis. Another symptom existed also to a marked degree, which has been the subject of debate and experimental research in its relations to cerebellar lesions, viz. constant priapism.

The progress of the case can be summed up in a few words. The reeling gait gave way gradually to a helpless, paralysed, bed-ridden state; the sight failure passed into total blindness; complete deafness soon followed, and the hebetude passed into a condition of complete unconsciousness. A high fever (102° – 106°) supervened; and when this had persisted for about a week, without any lung or other inflammation to account for it, coma and death ended the scene, thirteen weeks after the admission of the patient. For several hours before death, "Cheyne-Stokes" respiration was noticed.

An autopsy was held, of which the following are the notes:—Heart, liver, kidneys, spleen, normal; some pulmonary con-

gestion. On opening the skull, the veins over the vertex of each hemisphere were seen, dark and distended, lying, like earth-worms in appearance, over the sulci. The convolutions of all parts of the cerebrum were very markedly flattened against the skull. Membranes were slightly opalescent, but free. Lateral sinuses full of black clot. Sub-arachnoid fluid very abundant. Brain normal on section, excepting that the grey cortex was thin and very pale. The fourth ventricle was bulged and greatly enlarged by a tumour, the size of a bantam's egg, which lay filling the enormously dilated cavity. The walls of the ventricle were smooth and shining, and nowhere encroached upon by the growth, which was freely movable, and attached only to the roof of the ventricle by two delicate strands of membranous connections, conveying vessels, &c., to it. The tumour in appearance resembled a large purple mulberry; was very soft and gelatinous in consistence, breaking away readily before the touch. No visible changes existed on section of the pons, medulla, or cerebellum. The velum interpositum was thick, and the veins of Galen enlarged. The "iter e tertio ad quartum" was dilated to size of a quill, and the lateral ventricles enormously distended with fluid.

The tumour was carefully preserved and sent to Mr. J. Bland Sutton, of Middlesex Hospital, for examination. He reports as follows:—"It was impossible to obtain a microscopic section of the growth, as it fell to pieces on being cut, so that one was obliged to tease it out and examine it piecemeal. It is a villous growth, and agrees in structure with the fringes of the choroid plexuses of the ventricles. It has a basement membrane of well-defined connective tissue, with coils and loops of vessels entering into out-standing villi, crowned with spheroidal cells, squamous-looking in places."

I venture to call attention especially to the position, structure, and size of this tumour, in all of which particulars it is uncommon. Mr. Jacobson, in his edition of "Hilton on Rest and Pain," mentions a case very similar. He describes the tumour in his case as "papillose," its position as "in the floor of the fourth ventricle," and its size as "that of a small hazel nut." In my case, the growth was not "in the floor," but was free in the cavity of the ventricle, and was apparently ten or twelve times as large as in Mr. Jacobson's. The same authority refers to two "remarkably similar cases," given by Virchow. As in his case, so in mine, the effect of the growth was to close the cavity of the fourth ventricle, thus preventing the exit of fluid from the interior of the brain. The tumour, in the case of which I write, was so large that it completely blocked the cavity of the fourth ventricle, and thus prevented fluid from

the laterals from escaping, when necessary, through the "iter e tertio ad quartum," which was itself dilated largely, as far as its lower opening, a fact which evidences the amount of pressure above. The accumulation of fluid in the cerebral ventricles was also perhaps increased and hastened by the obstruction afforded by the tumour to the return of blood from them along Galen's veins. Great distension of the ventricles resulted, and hence the compression of the brain outwards against the skull, which caused the dementia and ultimate coma. The cerebellar symptoms mentioned in the notes of the case may be accounted for by the fact, that the tumour pressed upon the middle lobe of the cerebellum, which was during life diagnosed as the seat of lesion. Resting as it did upon, and irritating the posterior parts of the medulla oblongata, it caused the constant priapism; Eckard has produced experimental evidence pointing to such a conclusion. The difference which existed in the hearing powers of the two sides is difficult to account for; doubtless, however, the deafness and the vomiting which occurred depended upon immediate irritation of the roots of the auditory and pneumogastric nerves in the floor of the fourth ventricle. The wonder is that a growth of such size could have existed where it was without causing earlier grave interference with the vital functions of the medulla; possibly it grew very slowly, and the medulla accommodated itself to circumstances. The very early sight failure is attributable to the direct pressure of the tumour upon the corpora quadrigemina.

The high pyrexia which occurred is interesting. No signs or post-mortem evidences existed of inflammation either within the skull or elsewhere. Its occurrence in cases of simple tumours within the skull is on previous record; and apparently the importance of the proximity of the growth in this case to the presumable site of the hypothetical heat-regulating centre is diminished by the occurrence of pyrexia in cases of intracranial tumours situated remote from the medulla. Be the heat-regulating centre where it may, does not the increase of the body-heat in cases such as this, unaccompanied by any discoverable lesion of an inflammatory nature, point forcibly to the importance of the nervous factor in the production of pyrexia?—to an inhibitory power exercised in health by the high centres over metabolism, the "paralysis" of which power allows tissue-change to run riot, and a rapid combustion to ensue, attended by high fever, and the wasting which, during the last stages of this boy's life, was surprising in its rapidity.

CASE OF CALCAREOUS GUMMA IN THE BRAIN.

BY G. LACY BARRITT, M.R.C.S., L.R.C.P. (LOND.),

Resident Assistant Medical Officer, Union Workhouse Infirmary, Crumpsall.

WM. GIBBONS, æt. 35. Admitted into hospital May 6th, 1885.

Family history, good.

Previous medical history, very good up to twelve years ago, when he contracted syphilis; since that time he has been troubled by secondary symptoms, and has been very intemperate.

Condition on Admission.—Patient was a well-nourished, muscular man, looking rather more than his actual age. Had a distinctly alcoholic appearance; and also showed signs of acquired syphilis, *i.e.* had a faint cicatrix on the glans penis; amygdaloid glands in both groins, and a copper-coloured cicatrix of an ulcer over the middle of the anterior surface of the left tibia.

Complained of having had fits for three weeks previous to admission, gradually getting more severe and frequent. Had also nocturnal headache for two or three years previous to the present attack, much increased in intensity during the three weeks previous to admission. Patient localised the pain to the right side of the head, over the parietal and frontal regions.

Patient had fits every quarter to half hour, lasting about two or three minutes. These fits were peculiar in character, exhibiting the following phenomena:—

When attacked, patient threw his head back suddenly from *spasm of the muscles at the back of the neck*; the chin being tilted to the left side. In a few seconds clonic spasms occurred in the muscles of the neck to the back and front, the head being jerked backwards and forwards. When this condition had lasted about half a minute, violent spasms occurred in the arms and legs on both sides of the body; the spasms arising simultaneously in arms and legs. Lastly, the muscles of the glottis were attacked, respiration being suspended for a few seconds. Immediately after this the patient regained consciousness, and was in full possession of all his faculties.

Patient was put on large doses of iodide of potassium; with a draught of chloral and bromide at night. The fits slightly decreased in frequency, but the patient became very weak and

exhausted, and died on May 11th, five days after admission. Temperature varied from $98^{\circ}6$ to $99^{\circ}4$.

Autopsy.—On opening the skull, the meninges were found much congested, the veins being greatly distended.

The membranes were adherent to the substance of the brain over the upper part of the parietal convolutions on the right side.

The brain itself was very deeply congested, numerous points of capillary hæmorrhage being seen.

The lateral ventricles each contained a small calcareous nodule, imbedded in the choroid plexus.

Occupying the substance of the right temporo-sphenoidal lobes, was found a very hard mass about the size of a large walnut; the brain substance around the mass being very deeply congested. Section of the tumour showed it to be a cyst cavity, most probably gummatous, with calcified walls about $\frac{1}{4}$ in. thick. The contents of the cyst consisted of oil-globules and granular detritus, with numerous cholesterin crystals.

There was no further trace of tertiary syphilis in the body; but all the arteries showed commencing calcareous degeneration.

Remarks.—The peculiarities of this case are--1. that the tumour, which must have been present for some years, should not have caused any grave symptoms till three weeks before death. 2. The suddenness of the attacks, there being no aura epileptica, and the equally sudden termination. 3. The spasmodic movements of the head and neck occurring some little time before any spasms occurred in the limbs.

Reviews and Notices of Books.

La Législation relative aux Aliénés en Angleterre et en Écosse.
Rapport des Missions remplies en 1881 et 1883. Par le
Docteur A. FOVILLE, Inspecteur général des Services Ad-
ministratifs au Ministère de l'intérieur. Paris. Baillière
et fils, 1885.

THIS excellent *précis* of the English Lunacy Laws might well have been its funereal *éloge*, but "comes the accursed Budget with its shears, and slits the thin spun life" of the old Government, and withal of the new law; not to the advantage of any one, perhaps, in this particular regard, for the existing law relating to lunatics in England is irrevocably condemned; and it is not probable that the penalty it must eventually undergo for its blunders and misdeeds will be mitigated by delay. However, it is not our present purpose to venture, even in imagination, into the undiscovered country of the legislative future of England. Our task is to give some more or less adequate account of a report upon the lunacy laws of this very definitely discovered country by a great French official, and to appreciate the fair and logical criticism with which it is interspersed, to give a *résumé* of a *résumé*, an essence of an extract, if it so may be, without becoming absolutely obscure, in the essential brevity to which we are reduced. A Commission was appointed by the French Government in 1881, having for its object the study of a projected revision of the French law of lunacy of 1838, and of this Commission, M. Foville, the Author of the present Report, was appointed a member, on account of his position as Inspector-General of the Administrative Services of the Interior—or of the Home Department—as we should say, and he wisely took advantage of the opportunities afforded by the Medical Congress of the same year, which met in London, and of the Medico-Psychological section of that Congress, of which he was one of the Vice-Presidents. Perhaps the facilities for enquiry which he was thus able to enjoy may to some considerable extent account for the wide and sound knowledge which he has acquired on the complicated subject of his investigations, but the excellence of the work he has done can only be attributed to the author's own mental qualification for a task, simple enough in its design, though extremely difficult in its satisfactory accomplishment. For this *précis* is not only precise; it is, considering the limited space within which it is condensed (little more than 200 folio pages) extraordinarily complete; and it is so accurate, that its rare inaccuracies are of that recondite kind that they would scarcely be discovered by any one not actually engaged or

interested in the actual administration of the law. We think that there is an inaccuracy of this kind at page 31, where the Author says that a patient (*une malade*, he has missed our expressive term "alleged lunatic") having made a demand that his mental state shall be decided by a jury, the demand must be complied with, unless the Lord Chancellor, having personally examined the patient, has recognised the fact that the patient is unable to form and express the desire to be tried by a jury. This we believe is not so, seeing that the right to be tried by a jury is a common law right which the Lord Chancellor himself cannot refuse. That which the Lord Chancellor can and does virtually do, is to make the legal representatives of the alleged lunatic understand in these transactions, that "This demand for a jury not being the demand of the lunatic, who is not capable of making one, if it is persisted in, the costs of the inquisition will not be paid out of his estate." And as such a demand is in fact the demand of the solicitors for the purpose of increasing their professional emoluments, such a threat is almost always efficient. Another inaccuracy of a similar kind on this same subject is the assertion (page 32), that the jury in an inquisition must be composed of twelve members, unless the Lord Chancellor orders it to be more numerous. The law really is, that a concurrence of twelve jurymen is necessary to a valid verdict of unsoundness of mind, and a greater number than twelve is therefore impanelled, in order to increase the probability that twelve of them will agree to such a verdict. Again, there is a small inaccuracy of the same kind in the statement that the alleged lunatic is always visited and examined by one of the Lord Chancellor's Visitors, who makes a special report to the Lord Chancellor on the result, before the inquisition. The fact is, that the great majority of inquisitions are held without any preliminary official examination. The statute provides that the Lord Chancellor or the Lords Justices may send one of the Medical Visitors to ascertain and report whether an alleged lunatic is in a state of mind which enables him to form and express a desire to have a jury, and under this power the Lord Chancellor or the Lords Justices are able to obtain a report they can rely upon, in any case where a jury has been demanded. Before this statute was enacted in 1862, it was not easy for the Lord Chancellor to obtain an independent report on an alleged lunatic whose sanity was defended. Some independent physician was occasionally appointed a referee by the Court for the particular enquiry, and his sealed report was taken up by one or other of the litigating parties who chose to pay the fee for it, and who occasionally found that he had so paid for an opinion adverse to his cause. The present law is an improvement in the direction of simplicity, economy, and efficiency. But surely it is only an instalment, and the criticism of the French Commissioner on the complicated and costly procedure by which alone moderate fortunes can be protected on behalf of those who are obviously and unquestionably insane, cannot long remain foreign to the knowledge and the attention of the Legislature. Our French critic, so friendly and appreciative of all that is worthy of approval, points out that under our present law, whenever some person,

a helpless idiot from birth, perhaps, living in some remote corner of England or Wales, comes into possession of some small property which it is necessary to place under the protection of the law, by making the patient a Chancery lunatic, one of the Masters in Lunacy must transport himself a long distance, and proceed upon the spot to the formalities of an inquisition which never cost less to the lunatic than 1250 francs, the average untaxed costs being 2000 francs for undefended cases. Surely, he says, an examination and report from one of those officials in lunacy, whose function it is to travel personally all over the extent of the Lord Chancellor's jurisdiction, would justify a relegation of the central authority as to the formal legal declaration of incapacity to the tribunal which is nearest to the residence of the lunatic.

M. Foville is well aware of the provision which was made in 1862 for the protection without inquisition of the properties of lunatics which did not exceed a capital of one thousand pounds, or an income of fifty pounds a year, and he is also posted up to the last change of our law in 1882, whereby this limit was extended to double the above amount; but he remarks that the number of lunatics for which the first provision has been utilised is scarcely more than twenty, and it is not known to what extent the enlarged limit has been taken advantage of. It is, moreover, to be remarked that this form of protection without inquisition does not extend to the persons of the lunatics. The property is made available, but the lunatics themselves are neither visited nor protected. They may at present be called the Lord Chancellor's unprotected lunatics; but it is very likely that they will not remain so, now that the limits of the privilege are less restricted. M. Foville says that our proceedings are comprehensible as relating to certain doubtful and controvertible cases of insanity, or where considerable interests are involved, but he remarks that "The measures I have described [relating to Chancery lunatics] only apply to about 1000 insane persons, while the number of lunatics under treatment on the 1st of January, 1883, was 76,765. Has nothing then been done for the protection of the interests and the administration of the affairs of the 75,000 lunatics who are not under the direct and personal guardianship of the Lord Chancellor? Nothing, or next to nothing; and it is not one of the things which are the least astonishing to French alienists who study the Legislature of England, which has provided for the protection of great fortunes without preoccupying herself either with little ones, or the numerous interests of small importance which concern the indigent insane, which in France are attended to by administrative commissioners, or are under the surveillance of hospitals or asylums" (p. 39).

The Commissioners in Lunacy, remarks M. Foville, cannot habitually trouble themselves about the property of the insane who are under their protection. The only thing they can legally do is to report to the Lord Chancellor, when they learn that a lunatic is not treated in a manner corresponding with his supposed fortune. He says:

"The Commissioners have thought it their duty to interfere

with the financial interests of the insane, in order to indicate that which ought not to be done. In consequence of the impossibility of obtaining a regular administration of the property of lunatics without an inquisition, their relatives and friends have found themselves reduced to expedients to evade this difficulty; and for the purpose of effecting a remedy for frequent irregularities, the Commissioners in 1864 addressed a circular to the proprietors of asylums, desiring them not to give to their patients, under any circumstances, either the authority or the physical possibility of signing cheques, documents, or other papers, disposing of their possession or interest in their property."

In 1880, the Commissioners issued a new circular on the same subject, concluding with the following words, as translated from M. Foville: "The persons to whom lunatics are confided ought not only to refuse to their patients permission to transact business or to sign papers, but if they suspect the friends of their patients of the wish to obtain such signature secretly, they ought to act in such a manner that it cannot be done."

This action of the Commissioners elicits from M. Foville the remark, that it confirms the opinion he has expressed as to the insufficient protection afforded by the law of England for the property of lunatics, other than that of the small number of lunatics who have been found so by inquisition. He says:

"For all the seventy-five thousand other lunatics, one of two things must be true; either they possess absolutely nothing, and have no interests at stake, which is absolutely inadmissible; or their interests are either abandoned, or administered by the aid of subterfuges more or less irregular and blamable."

The dilemma is a very pretty one, and not to be avoided; but something may be said in mitigation of our logical critic's censure. The great bulk of small properties and of small businesses are capable of being administered without legal intervention. The wife or child carries them on for the insane husband or father by cash receipts and payments. More important interests are often managed for the insane under trusteeship, or by persons acting under the power of letters of attorney. On the whole, perhaps, we may say that this blot in our laws upon which M. Foville has put his finger, has not as yet created any great scandal. Its existence must be admitted, however, and if adequate investigation on the subject were to be made, it is highly probable that no little malversation of the property of lunatics would be discovered. But if the feeling of the public is as strong as it has been represented to be against the interference of the magistrate with regard to the personal liberty of an alleged lunatic, it is scarcely likely that the public sympathy would concur with any strong measures of judicial interference with every small property, interest, or business of every person insane, it may be, for a short time only.

Moreover the property of lunatics is protected generally by the law of the land to this extent, that fraudulent dealings therewith could in many instances be brought within the lash of the criminal law; and even those who dealt with it in a careless and irregular

manner, could be made responsible for damage and loss under the civil law. No doubt a lunatic is an unsafe and risky person either to transact business for or with, but he can neither be robbed nor cheated without grave risk. Neither is he absolutely free to repudiate any business engagements, on the ground that he was not of sound mind when he entered into them. If a man be so insane that he is not able to understand the nature of an engagement into which he enters, and the other contracting party knows it, the contract is voidable by law, and yet a good deal of business is transacted by people who are not in their right senses which could not be repudiated. The Courts will do justice, according to the merits of each particular case. Justice, however, is so costly, that for the poor it is too often unattainable.

And with regard to these circulars of the Commissioners in Lunacy, forbidding the proprietors of asylums from allowing their inmates to sign a cheque or to give a receipt, the good intentions of the Commissioners cannot be questioned; but it certainly may be questioned whether they were acting quite within the sphere of their proper jurisdiction when they issued these orders. A vast number of insane persons are confined in asylums who could not possibly be interdicted by the operation of proceedings in Chancery, persons slightly insane, or partially insane, against whom no jury would find a verdict that they were of unsound mind, and incapable of managing themselves and their affairs. It would seem to be a strong measure, and also one which the Commissioners have no power to enforce, to insist that no person of this kind shall sign a cheque for the support of his family, or a receipt for rent, interest, or dividend. Of course such acts may be of a questionable kind. They may even have the appearance of extortion, as when a person detained in a private asylum has been unable to obtain his liberty until he has consented to sign a cheque in discharge of claims for his maintenance therein; and no doubt there may be many other kinds of business transacted by insane persons in a manner equally open to censure. But there are many other kinds of routine business which may be transacted, both innocently and honestly, by persons suffering from certain phases of mental unsoundness, which would scarcely be upset by any court of law. That there are so few cases brought into the Courts, in which a remedy is sought for malversation of the property of lunatics, would seem to indicate that the blot in our law in this respect is much smaller than our French critic has been led to suppose. Whether it is sufficiently important to justify legislation, is a further question. It certainly is not sufficiently grave and pressing to need that all insane persons should be liable to interdiction, as M. Foville maintains, and as the circulars of the Commissioners seem to imply. That which perhaps is wanted is an independent judgment of the capacities and requirements in this regard of each particular lunatic who is under control; and if the Commissioners had insisted that no business of any kind should be transacted by any person confined in any of the asylums under their direct control, without the knowledge and consent of one of their own

body, their demand would have been more in accordance with the infinite variety of the circumstances and conditions of the insane, and with their own functions of regulation and protection. It is quite a different matter for the Commissioners to issue their fiat, or fiat, that no cheque or receipt shall be signed by any inmate of an asylum, from the refusal or permission of a Commissioner or of Commissioners cognizant of the circumstances in a particular instance.

And this remark leads to the criticisms in the Report we are examining, which are directed against the constitution and functions of the Board of Commissioners themselves (p. 53). While rendering full justice to the important results which the Commissioners have achieved by the vigorous impulses which they have given to their branch of public assistance, to the uniform regularity which they have introduced into a highly complex machinery of legislation, to the punctuality with which they have discharged their functions of surveillance, and to the balance they have maintained of firmness and courtesy which ought always to exist between persons exercising liberal professions, M. Foville proceeds to comment upon the present insufficiency of inspection.

"In this order of ideas is to be remarked the inequality which exists between the measures of surveillance taken in relation to those insane persons who are interdicted [that is to say, the Chancery Lunatics], and those who are not, the Visitors occupying themselves with the individual surveillance of the former with a method of extreme particularity, while the Commissioners are only able to devote to the second an attention which is superficial and of small efficiency."

"To this one adds that without doubt they [the Commissioners] are able to assure themselves of the general good appearance of the asylums, and to watch that legislation be observed in its entirety. But the details must of necessity escape them; they are not able to descend into the individual examination of the patients; the complaints of each of the patients in confinement are not studied with the care which they admit of; the aim of the law, which was to surround individual liberty with the most earnest guarantees, is not sufficiently attained" (p. 54).

The French Inspector-General makes further comments, that the visits of the Commissioners to the insane detained in workhouses are but accidental, and that the indigent insane under domestic treatment receive no visits from them at all; and that the Commissioners and the Visitors, discharging analogous functions, constitute two distinct Boards, to the evident waste of labour, time, and money, exclaiming, "*N'est-ce pas là un véritable gaspillage de travail, de temps, et de l'argent?*" With regard to these imperfections, M. Foville thinks that future legislation will be able to perfect the work of 1845, the results of which, however, have been too good to make it possible that it should be entirely reversed. No one, he thinks, is disposed to question the principle, that the surveillance of the insane should be exercised by the State; far from that, he says, the general conviction is that it should be more extended and

more particular, in order that it may afford a better guarantee of good service.

M. Foville concludes this chapter of his Report with a brief account of the parallel public service in Scotland, for which he has no adverse criticism, but which he makes to serve as a contrast to the defective system in this greater country. Concerning the guarantees for the protection of the insane, he says that the inspections of the Scotch Commissioners take the first rank, and that they are to be distinguished from the visits of the English Commissioners, in that while the English Commissioners make their visits in pairs—of a physician and a barrister—the Scotch Commissioners go alone, but they go often. And he concludes by a quotation from the address given by Dr. Lockart Robertson at the International Medical Congress.

“C'est seulement en Écosse que l'ensemble du service des aliénés du royaume se trouve réellement bien connu et bien contrôlé par le Bureau des Commissioners” (p. 60).

Whether the above opinions are well founded on the actual state of affairs in this country, most of our readers will be able to judge for themselves. For us thus much is certain, that this State paper from the great centre of Continental civilisation, from the country where the humane and scientific treatment of the insane had its origin, has dealt with our merits and defects in a manner which manifests a very remarkable knowledge of its most complex subject. It is quite evident that the reporter has met with ready help and instruction in his investigations, and indeed in his Preface he mentions many names of those to whom he recognises that his thanks are due in this respect; foremost among whom he names Dr. Hack Tuke, who, ever since the Medical Congress, when M. Foville's investigations commenced, “n'a cessé d'être, pour l'auteur, un guide et un auxiliaire d'une complaisance infatigable.” The report, however, has not been written for us, but for the instruction and guidance of the French Government, and it affords a striking contrast to the Blue-book on the lunacy legislation of foreign countries which has been issued by our own government during the past Session, supposably as a guide for our own legislation. This French Blue-book, or publication which corresponds with a Blue-book, is comprehensive, though condensed, critical, appreciative, admirably arranged, and on the whole very accurate. There is no publication we know of in the English language which affords, within moderate limits, so readable and reasonable an account of our lunacy laws and practice; while our own Blue-book on the lunacy legislation of foreign countries is a *rudis indigestaque moles*, or monument of consular methods how “not to do it.” Our criticism has naturally been directed to those parts of the publication in which our methods are most criticised, or to those parts with which we are not wholly in accord, and we have refrained, as unnecessary, to comment upon those portions of the work in which our lunacy laws have undergone a process of *précis* by a masterful hand. With matters of this kind we must assume that our readers are fairly well

acquainted from other and native sources of information, and yet for our own countrymen who wish to obtain a brief and precise, and a generally accurate view of our lunacy laws, we can strongly recommend a perusal of this foreign, but most well informed and appreciative exposition of them.

JOHN CHARLES BUCKNILL.

Etudes Cliniques sur la Grande Hystérie, ou Hystéro-épilepsie.

Par le Dr. PAUL RICHER. Second edition, 1 vol., 8vo., 975 pages, with 10 plates, and 197 figures. Paris: Delahaye, 1885.

We have given a full analysis of the first edition of Dr. Richer's excellent monograph in the January number of 'BRAIN,' 1882. As it now appears, the book does not only bear evidence of careful revision throughout, but is enlarged by about 250 pages of fresh material, and its pages are enriched with many new specimens of the author's powers as a draughtsman. Among the latter we observe a plate of etchings giving a synoptical view of over 80 figures, illustrating the four phases of the hystero-epileptic attack. With reference to the name hystero-epilepsy, it will be noticed that it is apt to convey a false impression. A point of cardinal importance in Professor Charcot's teaching is that the disease which goes under that name is not a combination of hysteria and epilepsy, but hysteria pure and simple, pushed to its fullest development. It is indeed impossible to define a limit between this "hysteria major" and the ordinary hysterical attack; the differences between the two are not qualitative, but quantitative. This subject is fully discussed and illustrated by Dr. Richer.

Typical cases of hystero-epilepsy are rare; of the four periods of the fit some may be ill-defined, or absent; others may predominate to such an extent as to overshadow all other manifestations. Hence a large number of varieties are observed, which is still further increased by the admixture of other elements. To the list of those described in his first edition, the author adds a variety with syncopal attacks, and two with exaggeration of spasmodic phenomena, noticed in the prodromal and in the fourth, or delirious, period respectively.

Dr. Richer has brought together into one chapter, now extending over 300 pages, a vast amount of most valuable information concerning hypnotic and allied phenomena among hysterical patients. A portion of it already existed in the first edition, and will be found analysed in our previous review. The physio-psychological importance of the facts now fully described by the author, and of the experimental results obtained by him and others, will be best realised on a perusal of his present account. Hystero-epileptic and hypnotisable subjects who present the phenomena of neuromuscular hyper-excitability, suggestion, and the like, have with much reason been called "the frogs of the experimental psycholo-

gist." The innermost springs of our mental constitution, the mechanism of our psychical life, are laid bare; and whilst so exposed to our view, they appear enlarged as under a microscope. In the normal state man thinks he is free, self-determined; but here, as Spinoza forcibly puts it, "he dreams with his eyes open." It is an illusion of the "inner sense," exactly as the existence of the outer-world, as we see and feel it, is an illusion of the external senses. In both cases consciousness is an epiphenomenon, not of the individual nerve-activities, but of the resultant of these activities. Hence, not being directly informed of these processes of manufacture, the human mind naturally falls into errors which only observation and experimentation can correct.

It is becoming daily more evident that the methods of physiological psychology, including psychophysics, must eventually occupy at least as important a place in science as the introspective and analytical methods hitherto almost solely employed by students of the mental processes. The comparative method too, from the evolutionist's point of view, is full of promise, though beset with danger from the natural tendency of man to create minds after his own image, and to endow mechanisms with consciousness. A corrective to this tendency is happily found in the experimental study of morbid psychology, through which the almost boundless expanse of the subconscious stratum of mental life is beginning to be revealed; and our debt of gratitude to Prof. Charcot for the impulse he has given to this department of science bids fair soon to equal that we owe him already as neuropathologists in the narrower sense.

The artistic powers of Dr. Richer have found a congenial field for their exercise in an Appendix ("L'hystérie dans l'art"), in which he reproduces some apicent delineations of the phenomena of so called "possessions." The historical and moral interest presented by the recent investigations into the question of hysteria scarcely yields to their importance from a medical standpoint.

A. DE WATTEVILLE.

A Treatise on the Diseases of the Nervous System. BY JAMES ROSS, M.D., LL.D., Senior Assistant Physician to the Manchester Royal Infirmary, &c. 2 vols. 8vo., pp. 1023 and 1047. London: Churchill, 1883.

WE reviewed the first edition of this great work in 'BRAIN,' January 1882. We may say at once of the second edition, which we heartily welcome, that it is even better than the first. There is no work dealing with neurological subjects which, so far as we can judge, is at the same time so practical and so philosophical. It is one which the advanced Neurologist will frequently consult, and one by aid of which the beginner of the special study of nervous diseases will be able to start well, that is, to start methodically.

Again, by it the practitioner will be helped to deal with particular cases of nervous disease, to recognise their real nature for medication and general care. We may condense the foregoing several statements by saying, that Dr. Ross has "made nervous diseases easier" to the scientific investigator, to the practitioner, and to the medical student. In particular we would commend this work to the young alienist physician, who, indeed, before he begins the special study of insanities, the most difficult of all diseases of the nervous system, should have a good knowledge of the comparatively simple diseases of this system. We say this, not only because Dr. Ross's book gives a full account of these simple diseases, but also on account of its great accuracy, clear method, and great insight. It is a very inspiring book; to read it carefully is a discipline, a training for wide and yet precise observation. Repeating from the former review our remark, that the book is too good for mere praise, we shall try to content ourselves for the rest by merely giving some account of the principal changes from the first edition.

In this edition the Author supplies full and definite references to the very many books and papers he has consulted. Much of the text is new, in the sense of being greatly revised and considerably altered. We now refer to particular changes. There are some changes of opinion as well as developments of views previously taken. The Author (I. p. 162), has given up the theory that tendon reactions are true reflexes, and has adopted Westphal and Waller's theory of direct action. The Article on Diseases of the Optic Commissure and Tracts is much altered, and almost re-written. Dr. Ross has abandoned Charcot's hypothesis of a supplementary crossing of the fibres of the tracts. The Anatomical and Physiological Introduction (p. 747) to Diseases of the Spinal Cord and Medulla Oblongata is rearranged, but not much altered. It is one of the most valuable parts of the work. The Chapter on Poliomyelopathies is considerably altered. Dr. Ross has given up the theory of the nervous origin of pseudo-hypertrophic paralysis, adopting the myopathic theory. Yet the disease is still described by him along with atrophic forms of paralysis, because of its clinical affinities, and for the sake of its discrimination from progressive muscular atrophy. Passing over much, we come to the second volume, which begins by a chapter, almost entirely re-written, on Progressive Locomotor Ataxy. The account given of this disease constitutes a valuable monograph; besides being well up to date as to what other observers have done, we have in it the Author's mature opinions on important questions still in dispute. We may here remark that in the narration of cases of disease of the spinal cord he has personally observed, the Author shows well the thoroughly practical side of himself; in quotations we gave in our review of the first edition, there was displayed his philosophical side. Both sides are well seen in the admirable Anatomical and Physiological Introduction to Diseases of the Encephalon. This introduction is, except for rearrangement, little different from that in the first edition. Passing on to the parts devoted to diseases of the encephalon, we especially note (p. 449),

as being very clear and most excellent, the "special consideration of Hemispasm, along with Paralysis, as it occurs in Infancy."

Dr. Ross, p. 498. quotes from 'BRAIN,' Vol. VI. 1883, p. 99, a case recorded by Dr. Thomason, of Oldham, in which conjugate deviation of the eyes (it had existed for four months) was removed by trephining. The patient was seen by Dr. Ross, who made observations on the boy's condition both before and after the operation. The case is a very remarkable one, quite unique, we believe. The Chapter on Neurasthenia is an original contribution to a subject which has been more written about than methodically studied. If this morbid affection is little serious in the sense of involving no direct danger to life, it is yet a very serious malady in the misery and incapacity it causes. It is an exceedingly complex disease, if disease we may call it. Dr. Ross's account of it is a very able one, and we think that it is so mainly because he comes to its consideration after having disciplined himself by careful and exact preliminary work on the simpler and, so to say, "coarser" diseases of the nervous system. This previous work enables him to make generalisations of this complex of symptoms after careful analysis. Hence we consider the statements he makes, from his personal observation of cases of this morbid affection, to be of more serious importance than those of many writers who have not worked at the nervous system "from the bottom upwards," and whose wide generalisations on so complex a subject as neurasthenia being made without prior analysis, are complete in a sense, but vague and non-realistic.

We have given sufficient illustrations to show the value of this edition, and in concluding our notice of it we can, without any misgivings, commend it strongly to all classes of our readers to the Student, the General Physician, and the Neurologist.

J. HUGHLINGS-JACKSON.

Lectures on the Diagnosis of Diseases of the Brain, delivered at University College Hospital, by W. R. GOWERS, M.D., F.R.C.P., Assistant Professor of Chemical Medicine in University College, Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic. London: J. and A. Churchill, 1885, pp. 246.

This work consists of eighteen Lectures, of which the first three are devoted to the medical anatomy of the brain. The arduous task of presenting shortly but clearly the present position of this question has been admirably performed, and the reader finds himself in presence of a picture which in its way is unique. The anatomy is constantly considered from a physiological standpoint, and in strict relation with the object of the book. Lectures 4, 5, 6, 7, and 8, are devoted to the consideration of motor, sensory

and cranial nerve-symptoms. Lecture 9 to mental symptoms. Affections of speech are discussed in the 10th Lecture. Various symptoms—such as headache, vertigo, vomiting, ophthalmoscopic changes—in the 11th and 12th. The remaining Lectures are devoted to the diagnosis of the seat of the disease, localization, diagnosis of the nature of the lesion, diagnostic pathology, and pathological diagnosis.

It is unnecessary to criticise the matter of this work. The reputation of the author is a sufficient guarantee of its accuracy. As regards the manner in which Dr. Gowers has performed his task, it may safely be said, we think, that the work is as well done as could be. It meets a distinct want which no existing book supplies. Indeed it would be necessary to cull from a large number of books in order to collect anything like the amount of information which is to be found in this work. Of necessity, in order to bring the subject-matter into the compass of a moderate-sized volume, the composition is extremely condensed; and the book is therefore not easy reading. Those who open it with the idea of getting a short cut to acquaintance with the most intricate of all problems in practical medicine, will be disappointed. It presents no superficial glimpse of the subject, but every page is full of advanced and solid instruction, which requires and deserves the most careful and repeated study.

T. BUZZARD.

Abstracts of British and Foreign Journals.

Pitres and Vaillard on Peripheral Neuritis. (*Archives de Physiologie*, Nos. 1 and 2, 1885, pp. 107-208).—Two further instalments of the valuable researches which are being made by MM. Pitres and Vaillard into the pathology of peripheral nerves, and of which Abstracts appeared in a former number of this Journal (Vol. VI. p. 555) are before us in papers entitled “Contribution à l'étude des gangrènes massives des membres d'origine neuritique,” and “Altérations des nerfs périphériques dans deux cas de maux perforants plantaires, et dans quelques autres formes de lésions trophiques des pieds.” The Authors have recently had the opportunity of making post-mortem examinations in two cases of extensive gangrene of the feet and legs, where there was no disease of the heart or blood-vessels to account for it, but in which, nevertheless, marked changes, amounting to almost total degeneration, were found in the nerves of the affected parts. One case was that of a young girl, the subject of chronic brain-disease, in whom, after years of suffering and increasing imbecility, and without any apparent cause to determine it, gangrene appeared in the feet, ran a rapidly destructive course, and, after sloughing patches had arisen in other and various parts of the body, ended fatally. Leaving aside the pathological changes found in the brain, of which full account is given in their papers, and which have no immediate bearing on the state of the nerves, there was extensive sclerosis in the antero-lateral and posterior columns of the cord, and in the nerve-trunks immediately contiguous to the gangrenous areas were found those degenerative changes which were described in the former Abstract. Nerves in other parts of the body were found quite healthy, and so also were the spinal roots. Their second case was that of an elderly woman who for some months had suffered from an unusual sense of fatigue in the legs, with more or less anaesthesia in the soles of her feet. Then suddenly her feet became cedematous, passed into symmetrical gangrene, and in a few days the patient died. Like changes were found here also in the peripheral nerves, but in neither case was any disease in the organs of circulation or sign of obstruction in the vessels discover-

able to account for the gangrene. The authors therefore conclude that the peripheral neuritis was the determining, though not perhaps the immediate cause of the gangrene. It is obvious, at any rate, that this peripheral neuritis is a more satisfactory explanation of those cases of spontaneous symmetrical gangrene which come on in the absence of vascular disease than is offered by the theory which ascribes it to some functional vaso-motor disturbance whereby a permanent contraction of the capillaries is induced and the parts are starved of blood. The authors ask whether the symmetrical gangrene in Raynaud's well-known cases, in which no examination of the nerve-trunks appears to have been made, may not also have been due to peripheral neuritis: and they point out how many events in their histories warrant the suspicion of some preceding nerve permanent condisturbance and degeneration. Such, for example, were the pains in the limbs, the occasional bullæ followed by ulceration, desquamation, smoothness or sclerodermic state of the skin, and the fall or dystrophy of the nails, phenomena which frequently persisted for a long time before the appearance of the gangrene, and which can have been but little dependent on simple vaso-motor disturbance, which is at best an unproved theory. They refer to the recent observations of Déjérine and Leboir (also recorded in 'BRAIN,' Vol. VI.) in support of their own views. Little less important, however, than the examination of the two cases which are the chief text of their paper is that made of the peripheral nerves in a case where the gangrene was unquestionably due to embolic plugging of the main vessel of the affected limb. Here the nerves were found perfectly normal, and it seems safe, therefore, to conclude that the neuritis is not itself induced by the gangrenous inflammation in the immediate vicinity of the nerves affected, a view which might perhaps have been entertained in the absence of this established fact.

The second contribution on the peripheral nerve changes in cases of perforating ulcer, so-called, contains less that is new, but is nevertheless a valuable addition to our knowledge of this disease. The paper deals especially with the changes which are met with at a distance from the seat of the ulcer, for the Authors have examined the nerve-trunks throughout their whole course, and thus discovered well-marked changes even in the sciatic itself, though less in degree than in more peripheral parts. In one of their cases degenerative changes were found in several other nerves than those leading to the perforating ulcer, in whose area of distribution, however, no so-called trophic changes had

arisen. The existence of a peripheral neuritis is evidently therefore not of itself sufficient to determine the "ulcer," and some other causes must be at work. What these are it is difficult to say, although no reference is made to the view, that continuous pressure on parts deprived of natural nerve-supply may have a potent influence in causing local ulceration, and allowing of its spread when once it has been started. A large part of this paper is devoted to meeting the objection raised by Michaud to the nerve origin of this disease. In a case recorded by him, Michaud had found degenerative changes in the nerves immediately adjoining the "perforating ulcer," but regarded them as of no significance in relation to the local sore, because he had met with precisely similar changes in the nerves of patients having no perforating ulcer at all. MM. Pitres and Vaillard admit that changes are by no means uncommon in the extreme periphery of the nerves of the toes, but in their experience they have been found only in cases where there have been at the same time such alterations in the skin or nails as really amount to trophic disturbance. It is by no means true that degenerative changes are constantly present, for in healthy adults and in young children the nerves are invariably quite normal. In addition to the cases of perforating ulcer, the Authors briefly record several other cases where there were so-called trophic changes in the nails and skin, and where undoubted degeneration was present in the terminal branches of the nerves in the affected parts, and they believe that this is a very common association. It must, however, be admitted that "trophic" changes cover a very wide field, and that all sorts of conditions seem to be included in the phrase. Coloured plates of the pathological changes in the nerves examined accompany both papers; of the first of which it may be said that it offers, if not an entirely new, at any rate a more probable explanation than has hitherto been afforded of symmetrical gangrene. Thus light is thrown upon such a condition as that recently recorded by Dr. Finlayson, of Glasgow, in the *Medical Chronicle* of January, where symmetrical gangrene of the extremities—fingers and toes—occurred as the closing scene of a case of very wide-spread scleroderma adultorum, and where no other disease was found post-mortem than "pulmonary tuberculosis," and nothing whatever to account for the gangrene. The history of the case shows that there had been considerable sensory disturbance for some time in the hands and feet, and the researches of MM. Pitres and Vaillard seem to supply the desired explanation of the various phenomena. Scleroderma has, it is true, been

often looked upon as the cutaneous expression of some nerve disease, but here we have a case in which symmetrical gangrene was superadded to it, and if the one condition find a real cause for its origin and progress in peripheral neuritis, it seems more than probable that the cause of the other condition lies also in the same morbid process.

HERBERT W. PAGE.

Anatomo-Clinical Researches on Bilateral Sclerosis consecutive to Unilateral Lesions of the Brain. PITRES (*Archives de Physiologie*, Feb. 15, 1884, p. 142).

A series of ten cases of hemiplegia is given, in which there was found, post-mortem, an *unilateral* lesion of the cerebrum with descending sclerosis in *both* lateral columns of the cord.

a. The duration of the cerebral lesion varied from five months to six years; the position was—twice in the cortex (motor area), seven times in the central part of the brain (especially middle part of internal capsule), once both the cortex and the central part was involved. The lesions were sometimes on the right, sometimes on the left side. Their character was—seven times old hæmorrhage, three times yellow softening (generally of moderate extent). There was therefore nothing special in their character or position to account for the peculiar bilateral degeneration of the cord.

β. At the level of the peduncles, pons, and medulla, there was the usual degeneration of the pyramidal tract, viz. on one side only, the same side as the cerebral lesion.

γ. From the level of the decussation of the pyramids downwards, there was degeneration in *both* lateral columns of the cord. In four cases the lateral column of the hemiplegic side (side opposite the cerebral lesion) was more markedly degenerated than that of the sound side. Upon the hemiplegic side there was what the author calls “massive sclerosis,” i.e. dense fibroid tissue with scanty nuclei, permeated by thick-walled vessels, and containing very few nerve fibres; upon the other side the sclerosis was “reticulated,” i.e. the fibroid tissue formed meshes of variable size, containing from one to twelve healthy fibres.

In six cases the degeneration was symmetrical. In these cases the area of degeneration extended further forwards than the usual area of the crossed pyramidal tract, and the degeneration itself was less dense and massive than is usual in unilateral descending degeneration.

The cerebellar tracts were unaffected.

The columns of Türek (direct pyramidal tracts) were normal in four cases, and in six slightly altered. There appeared to be no relation between the sclerosis of the columns of Türek and that of the lateral columns; thus in the six cases of symmetrical bilateral sclerosis, Türek's columns were twice symmetrically sclerosed, twice unequally sclerosed, once sclerosed on one side only: while in the four cases of asymmetrical bilateral sclerosis, Türek's columns were once healthy, once sclerosed on one side only, and that on the same side as the most marked lateral sclerosis (hemiplegic side).

Rest of white substance and grey substance was practically normal.

This bilateral descending sclerosis may be more frequent than is generally thought, for in forty cases of old hemiplegia, Pitres has met with ten examples of it.

Possible explanations :

(*a*) Charcot's hypothesis, that as the crossed pyramidal tract passes down the cord, it undergoes a second decussation, fibres passing from it through the anterior commissure to the corresponding tract of the opposite side.

But in the first place, Pitres could find no degeneration in the anterior commissure; and secondly, the bilateral sclerosis was developed, not gradually as the sections passed down the cord, but suddenly at the level of the decussation of the pyramids.

(*β*) That at the region of their decussation, where the two pyramidal tracts are in close anatomical relation to each other, a diffusion of the lesion takes place from the diseased to the healthy tract, giving rise to secondary descending degeneration in the latter.

Pitres inclines, on theoretical grounds, to reject this hypothesis, and he could find no anatomical evidence for it.

(*γ*) The author's own hypothesis is as follows :

That in addition to the variations which are at present known in the decussation of the pyramids, viz. that each pyramidal tract may at the decussation pass either wholly into the crossed pyramidal tract of the cord, or partly into this, partly into the direct pyramidal tract, and in a variable proportion into either, another variation is possible, viz. the pyramidal tract may here pass partly into the pyramidal tract of the *opposite lateral column*, partly into that of the *lateral column of the same side*.

As regards the symptomatology.

Diminution of gross muscular force on the non-paralysed side is

too common an occurrence in hemiplegia to be connected with this bilateral sclerosis.

Bilateral exaggeration of tendon-reflex does not necessarily accompany the bilateral sclerosis, and may occur independently of it. This the author's observations, here and elsewhere, show.

Double ankle-clonus has not as yet been shown, anatomically, to be connected with it.

Contracture of both lower limbs only occurred twice in the ten cases; and in one case (elsewhere published) of double contracture there was only unilateral sclerosis.

These facts are certainly not what we should expect, but we must remember that the spastic phenomena are only *indirectly* connected with lateral sclerosis: being due probably to the irritation of the anterior cornual cells set up by the lateral sclerosis.

The only symptom which Pitres thinks can be as yet connected with the double lateral degeneration, is a certain loss of equilibrium, a difficulty in standing and walking, which is experienced by some hemiplegics, who so far as the muscular power of the paralysed leg is concerned ought to be able to walk well. It is, he thinks, a want of co-ordinating power for the complex muscular movements required for these acts which need the combined action of the muscles on both sides of the body.

On the relations between Ankle-clonus and Knee-jerk. FLEURY (*Revue de Médecine*, August 1884, p. 656).—The author finds that the patellar tendon-reflexes are not always exaggerated in cases where ankle-clonus is present. In five cases observed by him there was ankle-clonus, but the patellar tendon-reflexes were normal. In three others, there was ankle clonus, but the patellar tendon-reflexes were feeble or absent. Further, in five cases of nervous disease where there was both exaggeration of patellar tendon-reflex and ankle-clonus, he found that by applying Esmarch's bandage the ankle-clonus could be removed without modifying the patellar tendon-reflex; hence the two phenomena cannot depend on the same cause.

J. A. ORMEROD, M.D.

B R A I N .

JANUARY, 1886.

Original Articles.

FURTHER OBSERVATIONS ON ALCOHOLIC PARALYSIS.

BY J. DRESCHFELD, M.D., F.R.C.P.,

*Professor of Pathology, Victoria University ; Physician, Royal Infirmary,
Manchester.*

SINCE my last communication¹ the subject of Alcoholic Paralysis has received a great deal of attention from different observers. In this country Hadden² has described two fatal cases, in one of which a complete autopsy was made :—in France a thesis by Oettinger³ has been published, treating fully of the history, symptoms and pathological anatomy of alcoholic paralysis ; and another thesis, treating more of those cases which simulate locomotor ataxy, is about to be published. In Germany several papers on this subject have appeared. The more important are by Löwenfeld,⁴ Strümpell,⁵ Knicke,⁶ Lilienfeld,⁷ and Schulz,⁸ who gives an almost full analysis of the hitherto published fatal cases. Of American neurologists, Hun⁹ has contributed one paper containing the report of two cases, one of which ended fatally.

¹ 'BRAIN,' July, 1884.

² 'Path. Trans.,' 1885, p. 49.

³ 'Étude sur les paralysies alcooliques,' Paris, 1885.

⁴ 'Arch. f. Psychiatrie,' xv. p. 438.

⁵ 'Berl. Klin. Wochenschr.,' 1885, No. 32.

⁶ 'Deutsch. Medicinal-Zeitung,' 1884, No. 72.

⁷ 'Berl. Klin. Wochenschr.' No. 45, 1885.

⁸ 'Neurol. Centralb.,' 1885, Nos. 19, 20 and 21.

⁹ 'American Journal of Med. Science,' April, 1885.

Within the last twelve months I have seen quite an unusually large number of cases of alcoholic paralysis, and several others have come under the notice of my colleagues at the Manchester Infirmary. Considering the widespread attention which this subject has attracted, it may not be out of place to briefly relate some of these cases, and to touch upon some points relating to the history, symptomatology, and prognosis of alcoholic paralysis.

Concerning the history of alcoholic paralysis I find that Magnus Huss¹ has been credited with having given the first description of a paralytic form of chronic alcoholism. On looking up the literature of the subject, I found, however, that an American writer, Dr. James Jackson, had as early as 1822-3 given a very succinct and at the same time very accurate and complete account of alcoholic paralysis.² The affection itself, the author names from the most prominent symptom arthrodynia; he states that he chiefly observed the disease among females, and that it is produced by "ardent spirits."

Dr. Jackson not only describes the pain, the paralysis and atrophy, but also gives an account of the "glossy skin"—a trophic change well marked in some of the cases recently seen by myself, and which has also been observed by others.³ Dr. Jackson's description of the disease is worth quoting, especially as the original is not very accessible. "This arthrodynia comes on gradually. It commences with pain in the lower limbs, but especially in the feet; and afterwards extends to the hands and arms. The hands may be affected first in some instances; and in all cases in an advanced state, the pain is more severe in the feet and hands, than in the upper parts of the limbs. The pain is excruciating, but varies in degree at different times. It is accompanied by a distressing feeling of *numbness*. After the disease has continued a short time, there take place some *contractions* of the *fingers and toes* and *inability to use these parts freely*. At length the hands and

¹ 'Alcoholismus chronicus,' 1852.

² The paper is contained in the 'New England Journal of Medicine and Surgery;' Boston, vol. xi. (third series: vol. i. No. 4,) 1822, p. 351 "On a peculiar Disease resulting from the use of Ardent Spirits."

³ Oettinger, p. 37.

feet become nearly useless, the *flexor muscles manifesting*, as in other diseases, *greater power than the extensors*. The whole body diminishes in size, unless it be the abdomen, but the face does not exhibit the appearance of emaciation common to many visceral diseases. The *diminution is especially observable in the feet and hands*; and at some time *the skin of these parts acquires a peculiar appearance*. The same appearance is noticed, in a slighter degree, in the skin of other parts. This appearance consists in a *great smoothness and shining*, with a sort of fineness of the skin. The integuments look as if tight and stretched without rugæ or wrinkles; somewhat as when the subjacent parts are swollen, but the skin is not discoloured. Yet in this disease there is not any effusion under the skin, and the character which this assumes, arises from some change in the organ itself." . . . (The *italics* are my own.)

The pains, present in the pseudo-ataxic form, have likewise been noticed by this accurate observer, for he says, "The most characteristic symptoms are manifested in the limbs; but the pain is not limited to these, and other symptoms are exhibited in other parts. The pain sometimes *shoots suddenly up one or both legs*, and in one case it frequently passed up the back and then forward to the pit of the stomach, taking the course of the diaphragm."

According to the more prominent symptoms, I have divided clinically this peculiar affection into two groups: the alcoholic ataxia, and the alcoholic paralysis. This classification has been adopted also by several authors—Löwenfeld, Moeli, Schulz, Strümpell and Krücke—who speaks of it as "Pseudo-tabes of alcoholics."

In considering the ataxic form, we have to distinguish between those cases where there is marked inco-ordination without much paralysis, and those where the gait resembles the ataxic gait, but is in some measure due to the paralysis of muscles of the lower extremity. The ataxic form represents a milder type; pathologically, however, as is evident from the cases reported by Déjérine,¹ Moeli,² and the case given below it is the same affection, namely, a peripheric neuritis.

¹ 'Arch. de Phys. norm. et path.,' 1884, No. 2.

² 'Charité Annalen,' 1884.

Of the purely ataxic form I have observed three cases, two of which have quite recovered, whilst the third died from uræmia, due to a contracted, gouty kidney.

The *first* case was a man, æt. 32, whom I saw with Mr. Fitzmaurice, of Bury. The patient had been for some years a heavy drinker, and had also indulged, to an extraordinary and almost morbid degree, in venereal excesses, but has never had syphilis. He complained of severe, lancinating and shooting pains in the lower extremities, sometimes in the upper extremities; both lower extremities showed some spots of anæsthesia and of retarded sensibility; the muscles of the calf were painful on pressure; there was no atrophy or paralysis of either upper or lower extremity, but there was marked inco-ordination. With his eyes open the patient could walk fairly well, and lifted the feet well from the ground, the heels coming down first; with his eyes shut, he showed marked ataxia; there was also distinct inco-ordination for finer movements in the upper extremities; the tendon reflexes were absent; no Argyll-Robertson symptom; no ophthalmoscopic changes. The patient gave up the use of alcohol, and completely recovered; and Mr. Fitzmaurice informs me that the tendon-reflexes, which remained absent for some time after the recovery seemed complete, have now returned.

The *second* case is a patient who is a boarder at the Cheadle Asylum, suffering from Dipsomania, and whom, in my capacity as one of the visiting physicians to that Institution, I had an opportunity of seeing frequently. For the notes, I am indebted to Mr. Reynolds, M.B., one of the assistant medical officers.

The patient, æt. 33, single, had an attack of sunstroke at Ceylon when 26 years old, and after that became very nervous and took to alcohol. He came to England, stayed some time, and drank very heavily; went back to Ceylon, and finally returned in 1882. He went to a Home for Inebriates, then to Loxley Hall, and finally came to Cheadle. He has never had syphilis.

His brothers are heavy drinkers, and one of them suffers from epileptic mania. In May 1884, he commenced to complain of pains in both legs and feet, especially on the dorsal surface of the feet near the toes, which were swollen; the pains were

shooting in character. There was hyperæsthesia of the skin over these parts, and some spots of anæsthesia; he had also pains in his fingers. He became very irritable in temper, lost his memory for recent events, and refused his food. There was loss of tendon-reflex, some slight atrophy of both legs, and marked ataxic gait. There were no bladder or rectum symptoms; the atrophy increased, the pains, however, became less, and the patient walked better. Towards the end of September 1884, the pains ceased altogether; the atrophy, however, remained, yet the patient walked much better. At the beginning of March 1885, he had pains over the distribution of the left supra-orbital nerve; he again complained of pains in his legs, burning sensations in the soles of his feet, and cramp in his legs.

In May 1885, I saw the patient myself. I found him somewhat irritable; otherwise but little affected mentally, except that he had lost his memory for recent events.

The gait was feeble and slow; he walked with the help of one stick, but his gait was distinctly ataxic, and became more so when he attempted to walk with his eyes shut. There was some emaciation of the lower extremities, but no marked paralysis of the muscles; the movements of the extensors of the toes were, however, sluggish. There was some slight atrophy of the muscles of the back and also of the arms, but the patient could flex and extend both fingers and wrists very well.

The patient complained of shooting-pains down the legs to the toes, occurring in paroxysms, and leaving a numb feeling behind. Firm pressure on the soles of the feet and on the legs was very painful. The patient also noticed a cold feeling when touched by any object. (This symptom Mr. Reynolds had noticed on several occasions previously.)

The limbs showed no vaso-motor disturbances.

The tendon-reflexes were absent.

The galvanic reactions were taken by Mr. Reynolds.

The contractions were slow, and followed by fibrillar tremors.

Extensors of the foot . . .	ACC and AOC, with 10 cells.
	KCC, with 15 cells.

A very strong magneto-electric current was necessary to get contractions of the extensors of the toes and calf-muscles.

The muscles of the upper extremities reacted well.

There were no eye or bladder symptoms.

There was loss of appetite, craving for drink ; the tongue was furred ; there was morning vomiting and occasional hæmatemesis.

The bowels somewhat constipated. The liver and spleen were not found to be enlarged.

The pulse was feeble and irregular.

As the patient refused to eat unless he had some drink, he was allowed beer. For the pains he had morphia injections at night, which gave him great relief.

The patient soon began to improve, and again went to Loxley Hall. He has returned from there recently, and is now quite well. He has gained flesh, he has no pains, there is no ataxia, and he shows no abnormality whatever in walking. The tendon-reflexes are normal, and the patient is now only kept as a boarder at his own request. He has never discontinued to drink beer, but avoids all other stimulants.

The *third* case was a female patient, æt. 53, who was admitted into the Infirmary, complaining of pains of shooting character in her lower extremities. Three years ago she suffered from gout in her hands. For some time she has been troubled with paroxysms of pain in her legs, without any swelling of the toes or any of the joints ; she has also noticed that her gait was awkward, and that she could not walk well in the dark. On admission, it was found that she had a typical alcoholic appearance ; the skin was dry, there was a nodular swelling on the metacarpo-phalangeal joint of the left index-finger. The patient looked thin ; there was no œdema, and there was no marked paralysis of any of the muscles. She could walk, but felt weak on her legs ; her walk was ataxic, and it was impossible for her to stand or walk with her eyes shut. She could move her limbs freely when in bed ; the tendon-reflexes were absent ; there was some anæsthesia of the skin of the lower extremities ; but very marked hyperæsthesia of the muscles of the calf and of the muscles of the forearm ; pressure on these parts caused most excruciating pain. The heart was found hypertrophied ; the urine of sp. gr. 1010, profuse in quantity, contained albumen and granular casts ; there was anorexia with

vomiting. The pupils reacted well, the fundus of the eye was normal.

Some time after admission the patient became delirious; there was incessant vomiting for twelve hours, and then the patient had a convulsion and died.

At the post-mortem examination made by Dr Harris, Pathologist to the Infirmary, the kidneys were found small and granular; the left weighed $1\frac{1}{2}$ oz., the right 1 oz.; on section, the cortical substance was found very much diminished, and streaks of urate of sodium were seen passing to the medullary part. Microscopically, marked interstitial nephritis was seen, with extensive deposits of urate of sodium crystals in the renal tubes. The heart was very much hypertrophied, and weighed $13\frac{1}{2}$ oz.; the myocardium was healthy, the valves normal. The liver weighed 3 lbs., and microscopic examination showed marked amyloid changes, together with monolobular cirrhosis, the fibrous tissue being still of very embryonic type; the spleen weighed 11 oz. The brain was anæmic; the ventricles contained more fluid than usual; the membranes of the brain were healthy; the pia mater, however, slightly opaque. The pons, medulla, and spinal cord had a perfectly healthy appearance, and were of firm consistence. The muscles of the leg and forearm were thin and pale. The sciatic, the ant. crural, and the musculo-spiral nerves were preserved for microscopic examination, along with the cord, and some muscles from the back of the leg.

The spinal cord, examined carefully after having been hardened, was found perfectly normal in all its parts.

The sciatic nerve appeared thin, grayish, and was surrounded by a great deal of adipose tissue. Vertical sections showed, when treated with perosmic acid, and stained afterwards with picrocarmin, a moniliform appearance of the nerve tubes, due to breaking up of the myelin; the nuclei were increased, and there was also some interstitial cell infiltration. Transverse sections showed in some few places an increase in diameter of the axis-cylinder, and again the interstitial infiltration.

The muscles showed chiefly increase of the muscle nuclei and an interstitial deposit of small round cells, and in some few places the striation was not well marked.

This case, both in its clinical history and in the post-mortem appearances, resembles very closely the first of the two cases described by Déjérine¹ as peripheric nerve-tubes, and there can be no doubt that both the cases described by this author are cases of alcoholic ataxia. We omitted in our case to remove portions of the skin, and I regret very much not to be able to state anything about the condition of the subcutaneous nerves, which were found markedly affected in Déjérine's cases.

The following case, which was admitted into the Infirmary only a few days ago, shows the combination of alcoholic ataxia with alcoholic paralysis.

W. B., æt. 41, was admitted into the Infirmary Nov. 10, 1884. The patient had been very intemperate and has a distinctly alcoholic appearance. He has had rheumatic fever, and has lately been very much troubled with pains in his limbs. He stated that he has had three attacks like the present, but not so bad, from which he recovered, after rest and abstention from drink.

The patient looks strong and stout; some of the muscles feel flabby, but there is no marked atrophy, though paralysis of some of the muscles is distinct.

In the upper extremity there is marked paralysis of the extensors of the fingers and of the wrists on both sides; some of the other muscles (triceps, deltoid) are slightly affected; the flexors act very well. Such movements as the patient is able to carry out show some inco-ordination.

In the lower extremity the extensors of the toes and of the big toe are but slightly affected; the peronei on both sides, however, are considerably paralysed; the arch of the foot is flattened, and the inner border is raised, whilst abduction is impossible.

The patient is able to walk with some assistance, but his walk is ataxic; he keeps his legs apart, and has to look to the ground for fear of falling; he cannot walk with his eyes shut. Isolated movements with either of the lower extremities show equally marked inco-ordination. With the eyes shut the patient does not know exactly the position into which his limb is put.

¹ 'Arch. de Phys.,' 1884, p. 231.

The sensory phenomena are those of alcoholic paralysis. The patient complains of shooting pains in his legs. There is cutaneous anæsthesia in both upper and lower extremity, irregularly distributed, with extreme muscular hyperæsthesia. There is also great pain if the skin apart from the muscles is firmly grasped. The sense for temperature is affected in both legs; the patient rightly distinguishes a cold body, but contact with a hot body gives him a sensation which he compares to that of an electric shock. There is some analgesia, and the prick of a pin is felt only after some time.

The tendon-reflexes are absent; the superficial reflexes fairly normal.

The pupils are normal, and react to light and accommodation; the special sense-organs are normal.

The mental condition of the patient shows symptoms which are often found in cases of alcoholic paralysis, and which have been noticed by other observers, without, however, laying great stress on them. The patient answers questions rationally; his memory, however, is very defective, and he suffers from delusions. Thus he tells you that he gets up every day, goes into the next ward, and converses with the other patients, though, as a fact, he has never left the ward since his admission. When further pressed, he even gives a description of the ward, details his conversation with the patients with a minuteness and readiness which is astonishing. I have noticed exactly the same peculiarity of the mental state in some of the other cases to be presently described.

The condition of the other organs calls for no special notice. The heart-sounds are normal, but weak; the pulse 120, compressible; appetite fairly good; the liver is normal; urine free from albumen.

We have so far been able to take the electric reactions to the galvanic currents only once, and muscles showed degenerative changes.

Peronei of right side	.	.	KCC, with 30 cells.
			ACC „ 25 cells.

The other cases which I have as yet to describe belong to the typical class of alcoholic paralysis, characterised by

well-marked paralysis with atrophy, affecting chiefly the extensors of the fingers and toes.

The paralysis and atrophy sometimes come on very acutely, at others more slowly. When these cases come under observation, the patients are as a rule unable to stand or walk, and it is therefore not easy to make out whether the paralytic stage is here preceded by an ataxic stage. As the sensory phenomena in these cases are the same as in the first group, it is highly probable that where the paralysis comes on slowly, pseudo-ataxic symptoms, as in the case just given, precede it. Of this group I have within the last twelve months seen four cases, all females, two of which have quite recovered, one is beginning to improve, whilst the fourth, when last heard of, had as yet shown no signs of improvement. As the symptomatology of these cases is now sufficiently well known, I will very briefly relate the cases, only drawing attention to particular points.

CASE I.—A lady, æt. 25, whom I saw with Mr. Renshaw, of Altrincham, in August 1884.

The patient had freely partaken of spirits, and had a constant craving for stimulants.

She had complained of some pain for some time; the pains were soon followed by paralysis and atrophy of the extensors of both fingers and toes, with paresis of the other muscles, so that the patient could neither stand nor walk, and could raise herself only with difficulty from the recumbent posture. Cutaneous anæsthesia and muscular hyperæsthesia were well marked. Tendon-reflexes absent, superficial reflexes much diminished.

The patient exhibited the same peculiar delusions just referred to. When I saw her she told me that she could walk very well, that she had paid several visits that morning, and minutely described all she had seen and done when visiting her friends. When her helpless condition was pointed out to her, she became highly emotional, and burst into tears.

The patient was removed to a small cottage, and attended by a reliable nurse, so that she had no access to drink; she completely recovered in three months. Mr. Renshaw informs

me that she now walks very well, and that the tendon-reflexes have reappeared.

CASE II.—A lady, æt. 42, whom I saw with Mr. Kitchen in September, 1884. Her symptoms resembled those of Case I. The paralysis had come on rather more slowly, and the atrophy was much more marked, and affected the greater part of the body. This patient also had the same peculiar delusions. Though entirely confined to bed, she told us that she went out every afternoon for a walk to the seaside; that she sits on the sand at the seashore, watching the waves and the passing steamers. (Her residence was at least twenty miles away from the sea.)

It was astonishing to listen to the account she gave of her seaside rambles, which had every semblance of truth, and yet was entirely imaginary.

We advised the patient's removal, if possible, as we were sure that the patient, unknown to her husband, was still supplied with stimulants. Our suggestion was, however, not acted upon, and when last I heard of the patient, she was still in the same helpless state, and rather more emaciated.¹

CASE III.—E. C., æt. 33, seamstress, married, was admitted into the Infirmary, Dec. 3, 1884, suffering from typical alcoholic paralysis.

She had suffered from "rheumatic" pains at different times. Three months before admission, the pains in her arms and leg became much worse; her legs often "gave way" when she attempted to walk, and a corresponding weakness was noticed in her arms. The weakness increased to such an extent, that she was obliged to keep to her bed, and had to be fed, not being able to use her hands. She admitted having partaken of alcohol in large quantities.

The patient had a characteristic alcoholic appearance. The muscles of the upper and lower extremity felt flabby; the extensors of the fingers and toes were quite paralysed; the

¹ Since the above was written, I have heard from Mr. Kitchen that the patient has lately much improved, and is now able to walk with the help of sticks. She has, however, the same delusions still. She has entirely abstained from spirits, but takes several glasses of beer every day.

hands presented the characteristic drop-wrist; the feet, the paralytic club-foot; the flexor muscles of the fingers and toes were paretic; the arm and shoulder muscles were fairly strong. The tendon-reflexes were absent, the superficial reflexes normal. There was also cutaneous anæsthesia in the legs and fore-arms, with marked muscular hyperæsthesia on firm pressure. The sense of temperature was affected on the outer side of each leg; the patient was able to distinguish cold, but not heat. The paralysed muscles were atrophied to a much greater extent than the paretic muscles.

The patient complained of loss of memory, but otherwise her mental condition was normal. The right pupil was irregular from an anterior synechia.

The application of the electric current was very painful.

A very strong faradic current produced no contraction in the extensors of the hand; the flexors responded to a strong current, but not to a weak current. With the galvanic current directly applied to the muscles, there was found to be degenerative reaction in the extensors of the fingers, the extensor carpi ulnaris, the extensor communis digitorum, and extensor longus pollicis. Some of the flexor muscles also showed degenerative reaction.

The patient remained in the hospital till March 1885, when she left us somewhat improved; she has since then completely recovered. She has lately presented herself in the out-patients' department of the Infirmary, and no trace of any paralysis or atrophy could be discerned. The tendon-reflexes had reappeared.

CASE IV.—M. E., æt. 43, was admitted into the Infirmary on July 13, 1885, under Dr. Leech, who kindly transferred her to me. The patient admitted having partaken of stimulants (whiskey and gin), and gave the usual history of rheumatic pains, with which she had been troubled for some time, followed by weakness which had increased to such an extent that she was rendered quite helpless. On admission, her condition resembled very much that of Case III., except that the paralysis was much more extensive. The patient was very stout; the muscles flabby; she was unable to raise herself from the recum-

bent posture; hands and feet were flexed; there was marked wrist-drop, the elbows were bent, the knees drawn up. The shoulders and hips could be moved, but the movements were sluggish, and when passive movement was attempted, the patient screamed with pain. The tendon-reflexes were absent, the superficial reflexes not well marked.

There was again cutaneous anæsthesia for tactile impressions, with well-marked muscular hyperæsthesia, affecting upper and lower extremities, and to some extent also the muscles of the abdomen. The sense for temperature was normal.

The skin on the dorsal surface of the fingers of both hands had a marked glossy appearance.

The patient's mental condition is affected; she answers questions rationally, and shows no incoherency in her answers, but she has delusions. She fancies she is at home, &c.; she is very restless at nights, and sleeps but little; she is also very emotional.

The tongue is covered with thick white fur; there is anorexia, occasional vomiting, and constipation. The liver and spleen are normal in size.

To the faradic current some of the muscles (triceps and extensors of the fingers and toes) do not respond at all; some, *e.g.* the biceps, supinator, the interossei, contract with a current of medium strength.

The application of the electric current is so painful, that the galvanic reaction could only be tried in the most affected muscles, and here marked degenerative reaction was found, thus:—

Extensors of fingers (left)	. .	ACC = 15 cells.
		KCC = 25 ..
		AOC = 20 ..

The flexors also showed degenerative reaction, thus:—

Flexors of fingers (left)	. .	ACC = 30 cells.
		KCC = 40 ..
		AOC = 45 ..

The patient has, up to now, made some slight progress; the extensors of the fingers on the left side show some power, likewise the extensors of the toes on both sides. There are

now, however, very marked contractures of the elbows, shoulders and knees, with absence of deep reflexes. The contractures are more pronounced on the right than on the left side. There is also some slight swelling over the back of the right hand (the tenosynovitis of Gubler, as in lead palsy).

The muscular hyperæsthesia is better.

The mental condition of the patient has undergone marked improvement; she has now no more delusions, and has no recollection of her former delusions.

I have no wish to prolong this paper by any further remarks. The symptoms of the disease are so clear, that there is no difficulty in diagnosing the affection.

The peculiar form of delirium seems almost as characteristic, though by no means as constant, as the muscular hyperæsthesia. Glynn¹ has already drawn attention to it. It occurs in chronic alcoholism without paralysis; and Mr. Mould, Superintendent of the Cheadle Asylum, informs me that he has constantly met with it amongst dipsomaniaes.

As regards the pathology, we have now abundant evidence in the post-mortem appearances that alcoholic paralysis is a multiple peripheric neuritis. This view is again supported by the third case of alcoholic ataxia given above. For an analysis of the results of the post-mortem examinations in alcoholic paralysis, I must refer to Schulz.²

¹ 'Liverpool Medico-Chirurgical Journal,' July, 1883, p. 374.

² 'Neurol. Centralb.,' 1885, No. 21.

ON A CASE OF MULTIPLE SPINAL AND CEREBRAL TUMOURS (SARCOMATA), WITH A CONTRIBUTION TO THE PATHOLOGY OF SYRINGOMYELIA.

BY THOMAS HARRIS, M.D. (LOND.),

Pathologist to the Manchester Royal Infirmary; Assistant Lecturer and Demonstrator of Pathology in the Owens College.

THE following case came under my observation a few years ago when resident in the Manchester Royal Infirmary; and as I was able to watch the course of the disease for a period of eight months, I have considered it worth while to now put it on record, since it possesses many features, not only of clinical, but of pathological interest.

The patient was under the care of Dr. Morgan during the greater part of his stay in the Infirmary, and it is to him that I am indebted for his kind permission to publish it.

W. D., a married man, aged 38, was admitted at the end of July, 1881, to the Manchester Royal Infirmary, under the care of Dr. William Roberts, complaining of weakness of the left lower extremity with aching pains in both legs. He stated that up to the commencement of the present attack he had never had any serious illness. No history could be obtained of any syphilitic taint.

His illness commenced about eighteen months previously, with aching pains in the lower extremities; but these were more marked on the left than the right side; in the course of a few months the left leg became weaker than the right, and this soon was so marked that, according to his own description, "the left leg would occasionally slip from under him, and he would fall to the ground." It was not until about nine months since that he noticed the wasting of the muscles on the front of the left thigh, which at the time of his admission was such a marked feature. At no time had he

noticed any numbness of either of the lower extremities, neither has he had any bladder troubles.

On August 1st, 1881, we have the following note as to his "present state":—

Patient is 5 ft. 4 in. in height, of healthy but not robust appearance; he complains of aching pains in both lower extremities, but they are much worse in the left than the right leg, and are sometimes so severe as to necessitate a sleeping draught being administered; he also complains of weakness of the left lower extremity. He is able to walk short distances, but this causes fatigue; he walks with no special kind of gait, but slight dragging of the left leg is observed. On examination, the chief feature which strikes one is the excessive atrophy of the extensor muscles on the front of the left thigh; the other muscles of this extremity appear also wasted, but not to that extreme degree which the above extensors show. He does not complain of weakness of the right leg, but all the muscles of it appear more or less atrophied. Patellar tendon-reflex of the left leg almost abolished; that of the right present, possibly in excess. At this date we have no note of any cutaneous sensory disturbance. There are no bladder troubles.

On August 6th he was sent to the Convalescent Hospital at Cheadle, but there became much worse, first noticing that the right lower extremity became numb and gradually weaker; it also became a seat of aching pains, which were far more severe than they had ever been in the left leg. He had also difficulty in passing his water, having to strain to do so.

On September 26th he was returned to the Manchester Infirmary, this time under the care of Dr. Morgan.

On his return he was just able to walk, but required great assistance to do so. He took to his bed, to which he was ever afterwards confined.

On October 1st we have the following note:—He is now not only unable to walk, but to sit up in bed, being unable also to raise himself from the horizontal to the sitting posture.

He complains of great pains in the right leg; this is not of a shooting but of a continuously aching character. There appears to be no hyperæsthesia of the skin, but when the

muscles of the leg or thigh are grasped, it gives him great pain. There is considerable wasting of the muscles of both legs and thigh, but that of the left side is more marked than the right, there being very little of the quadriceps extensor of the left thigh remaining. He has now almost complete paraplegia, being unable to flex the thighs on the abdomen, nor can he move the legs at the knee-joints; he can just manage to move the feet at the ankles and the toes on either side.

The quadriceps extensor muscle of the right thigh responds to a weak faradic current, but requires a moderately strong galvanic one to give a response, the kathodal closure contraction occurring with a fewer number of cells than the anodal closure one.

The quadriceps of the left thigh gives no response to the strongest faradic current, but with the galvanic current a contraction is obtained with a powerful current on kathodal closure, but no response can be seen with the strongest current at anodal closure.

The tibialis anticus of either side responded readily to a weak faradic current, that of the right leg to a slightly weaker current than the muscle on the other side. With the galvanic current, the kathodal closure contraction took place with a weaker current than the anodal closure contraction; the kathodal closure contraction occurring with the same strength of current on either side, but the anodal closure contraction ensued with a somewhat weaker current in the case of the right tibialis anticus, than it did with the muscle of the left side.

There is no rigidity of the lower extremities; on the contrary, they are quite flaccid.

Patellar tendon-reflex of the left side almost, but not quite, abolished, that of the right side is now considerably increased.

He has a slight feeling of constriction round the abdomen.

There now exists cutaneous analgesia and anæsthesia over the front and outer side of the lower half of the left thigh; also another small patch of anæsthetic skin, not more than an inch in diameter, exists over the margin of the costal arch

in the right mammary line. No other affection of sensation can be found in any part.

No weakness of the upper extremities.

There is no deformity of the spinal column, and no tenderness is detected on pressure along the spine.

Once since his return from Cheadle the catheter had to be used, on account of retention of urine, but usually he is able to pass his water, though he has to strain to do so. No bedsores, and no joint affections. Temperature normal.

No affection of the circulation or respiration, and the functions of other organs are performed in a healthy manner.

Nov. 8th.—He is still unable to move his lower extremities any more than he could a month since, but the continuous pain, and that caused by grasping the muscles of the legs, have both disappeared. He is still unable to raise himself from the lying to the sitting posture. Patellar tendon-reflex of left side almost abolished. That of right side slightly in excess.

The whole of the left lower extremity has now become anæsthetic and analgesic, this extending above to the level of a line drawn through the umbilicus, on the lower part of the abdomen it does not quite extend to the middle line in front. Over the right lower extremity sensation is impaired, though there are no distinct patches of anæsthesia.

The patch of anæsthesia still exists over the costal margin on the right side, and remains much the same in extent as noted a month back.

For the last three weeks he has complained of a heavy sensation over the right frontal region, and about the same time he noticed numbness of the right half of the face.

There now exists marked cutaneous anæsthesia and analgesia over the right orbit. Over the cheek and lower jaw of the same side, there is less acute sensation than on the left side, but there is no complete loss of sensation.

The conjunctiva of the right eye is much injected, and there is a superficial ulceration of the cornea of that eye.

The right masseter and right temporal muscle are considerably atrophied.

There is no affection of taste on either side of the tongue.

No vomiting or vertigo is present.

Patient is very intelligent, and there appears to be no mental disturbance.

No optic neuritis is seen in the left eye; the fundus of the right cannot be seen, on account of the ulceration of the cornea.

No oculo-motor troubles; no difference in size of the pupils of the two sides. No facial paralysis.

Hearing good on both sides, but much better on the left than on the right side. No difficulty in swallowing; no morbid symptoms connected with the circulatory or respiratory systems. Urine free from albumen and sugar.

No stiffness or rigidity of the muscles of the neck.

No paralysis of the muscles of the tongue.

No affection of speech.

He is able to pass his urine more readily, not having to strain so much to do so. Appetite good; bowels constipated. Temperature normal.

No bedsores.

Nov. 21st.—He is now unable to move either toes or feet at the ankle joints.

Some rigidity of the right lower extremity has come on; while he lies with the left leg fully extended, the right is flexed at the hip and knee joints, and the hamstring muscles stand out in marked relief; we can bring this limb into the extended position, but doing so causes patient considerable pain at the knee.

The atrophy of the muscles of the lower extremities is becoming more marked; this is especially perceptible in the right limb, where the quadriceps extensor cruris has very much diminished in volume, and is now nearly as much wasted as that of the left side, very little muscle remaining on either side; the girth of the thigh, three inches above the upper margin of the patella, is ten inches on either side.

The atrophy of the muscles of the calf and those on the anterior aspect of the leg is now rather more marked on the right than the left side. No patellar tendon-reflex can now be obtained on either side; even on the right side, where it was so much in excess, it is now absent.

No epigastric or cremasteric reflex can be obtained, but the abdominal reflex is present.

The skin on the right lower extremity is almost entirely anæsthetic and analgesic, except that at one or two isolated spots he occasionally recognises the prick of a pin, but he is still able to recognise heat and cold when applied over this limb.

The patch of anæsthesia over the right costal margin is rather more extensive, but it is separated yet by a portion of sensitive integument from the anæsthetic part below.

The anæsthesia over the left lower extremity remains the same ; it does not extend higher than the level of the umbilicus, and not quite to the middle line on the lower part of the abdomen.

The anæsthesia over the distribution of the ophthalmic division of the 5th of the right side remains the same ; but there is in addition a small patch of anæsthesia immediately below the right eye, while the rest of the skin of the cheek is sensitive, though not so acutely so as on the left side. The cornea has become more lazy. There is no evidence of the involvement of any cranial nerve, except the 5th of the right side.

There is no optic neuritis.

He is very little troubled with headache ; there is no vertigo or sickness.

There is now complete retention of urine, and the catheter has to be constantly employed.

For the last ten days there has been a slight evening rise of temperature, while the morning has been normal. This has been constant during that time, but it has not reached a higher point than 100° F.

His general health remains fairly good.

The loss of sensation has now extended until the whole of the right lower extremity as well as the left is anæsthetic and analgesic, though occasionally he can feel the prick of a pin at a few spots over this surface, but these sensitive parts have no regular arrangement.

Dec. 4th.—The anæsthesia has now extended, until that over the right costal margin and over the right lower extremity have merged into one. The upper limit of the anæsthesia on the right side is a line drawn through the apex of the xiphoid cartilage, while on the left side it does not extend quite so

high, the upper limit being a point about two inches above the umbilicus.

The atrophy of all the muscles of the lower extremities is most marked, it being much the same on the two sides. There is now observed slight swelling of the left knee-joint, evidently due to effusion of fluid into the interior of the joint; the fluid appears limited to the joint and does not involve the surrounding subcutaneous tissues. There is no redness over the joint and no pain, and patient was unaware of the existence of the swelling. The left ankle-joint appears also a little swollen, but this is less marked than the swelling at the knee.

Retention of urine still exists; there is now also incontinence of fæces.

A small bed sore has appeared in the central line over the upper part of the sacrum.

Anæsthesia exists now over the whole of the right half of the face, except over the lower jaw. The ulceration of the right cornea and injection of the conjunctiva has increased.

The right half of the upper lip is swollen and thickened, and on the inner aspect is an ulcer the size of a sixpenny-piece; it causes him no pain, in fact he is not aware of it, merely noticing the swelling of the lip; opposite this ulcer there is rather a sharp rough tooth.

The palpebral fissure of the right side is smaller than the left. There is no evidence of any other cranial nerves being involved. He has complained lately of a little more pain over the right half of the face and right frontal region.

There appears to be distinct flattening of the left half of the chest below the nipple; the movement here is not so good as on the right side, the ribs appearing to be dragged up, but the chest undergoing very little expansile movement. There is no anæsthesia over this flattened part. Percussion and auscultation of the chest give negative results.

The temperature still shows slight evening rises.

Dec. 15th.—The limits of the anæsthesia of the lower half of the body remain stationary; the loss of sensation does not appear to be extending at all. We have a note at this date that the movement of the chest appears much the same on either side; if anything, it is deficient on both sides.

The retention of urine is beginning to give place to incontinence. The incontinence of fæces persists. The urine is acid when drawn off and contains only very little pus.

The temperature has lately shown more considerable variations; for the last three nights it has risen to above 101° ; last night it was $101^{\circ}\cdot6$, and the previous evening 102° ; it falls to normal in the morning.

There appear to be no local changes in the lungs to account for this pyrexia.

The condition of the right eyeball, as regards the neuro-paralytic ophthalmia, remains stationary; the palpebral fissure is still smaller on the right than the left side.

Another ulcer has now appeared, this being on the right half of the lower lip in a corresponding position to the one on the upper lip. The latter ulcer still exists, and neither ulcer extends across the middle line to the left.

No other cranial nerves besides the 5th are involved. There is no optic neuritis. He is now very little troubled with headache.

Jan. 10th, 1882.—His general condition has changed much for the worse; his appetite fails, and he has lost a great deal of flesh this last fortnight.

There is incontinence of urine, and the fluid is very purulent, alkaline, and offensive.

The temperature chart still shows considerable evening exacerbations.

The condition of the lower half of the body remains the same, except that the effusion into the left knee-joint has disappeared, and the joint has returned to its normal condition.

There is no loss of power or anæsthesia of the upper extremities.

The ulcers on the inner aspect of the lips still persist.

Jan. 25th.—Patient continues to get worse, he has emaciated very much lately.

The urine is very foetid and purulent, so the bladder is now washed out frequently with a solution of boracic acid.

There is still a considerable evening rise of temperature; about a week ago he had a shiver, and the thermometer re-

gistered $104\cdot4^{\circ}$, but this temperature was not maintained for many hours, it soon falling to $101\cdot4$.

The grasp of the left hand has now become distinctly weaker than the right, but the anæsthesia still maintains the limit it has done for so long, and does not extend.

The expansile movement of both sides of the chest appears deficient; auscultation and percussion of the chest give negative results.

The atrophy of the muscles above the knees appears the same on the two sides, but below the knees it is more marked on the right than left side, a difference which is especially marked in the calf muscles.

During the last few days a red erythematous patch, followed by the development of a bulla, has appeared over the external malleolus of the left leg; a similar formation has ensued on the under and inner aspect of the left heel.

The anæsthesia over the course of distribution of the 5th cranial nerve of the right side still exists, but a great change has ensued in the condition of the right eye. The conjunctival injection has entirely disappeared, and the eyeball appears quite pale, like the left eye; yet the cornea is still opaque.

For the last fortnight a small ulcer, about the size of a split pea, has existed on the skin of the lower eyelid, near the inner canthus, of the right eye.

The ulcers on the mucous surface of the lips have almost healed.

No optic neuritis nor evidence of the involvement of any cranial nerves, except the right 5th.

Feb. 4th.—Patient has complained these last few days of rather more headache.

There is still incontinence of urine, but the fluid has become of a clear amber colour, not at all offensive, acid, and contains no albumen.

The temperature is still a little elevated, but does not show such considerable variations as at the date of the last note.

The ulcers at the inner aspect of the lips and at the inner canthus have now healed.

The weakening of the left arm has become very marked,

the grasp of the left hand, as registered by the dynamometer, is 9, while that of the right is 45. Not only is the grasp enfeebled, but the power of movement at any of the joints of this extremity is much lessened. No anæsthesia is found over the arms; the upper limit of the loss of sensation remains the same.

The condition of the chest movement is the same as at the date of the last note, there is very little expansion movement of the lower part on either side.

The bed sore on the sacrum still exists, but does not increase in size.

The bulla over the left external malleolus has given place to a small sore; that under the left heel has dried up.

The skin along the outer side of the left foot has become erythematous, apparently due to pressure, the foot resting on this part. The skin under the right heel has become dark, soft, and rather pulpy, but it is not ulcerated.

Feb. 10th.—The condition of the left arm remains the same; the triceps tendon-reflex is increased on either side, that of the left distinctly more so than the right.

The chest movements remain the same. Not only below but also at the upper part of the chest there is very little expansile movement, and respiration appears to be chiefly diaphragmatic.

There is effusion into the right knee-joint, which is swollen, but not reddened or painful.

Feb. 18th.—The effusion into the right knee-joint still persists.

The conjunctiva of the right eye has now again become much injected.

Triceps tendon-reflex is still increased on either side.

Feb. 25th.—Condition of the left arm remains the same; there is no evidence of the involvement of the right upper limb.

The thoracic movement still remains very defective.

There is still slight effusion into the right knee-joint.

There has now appeared internal strabismus of the right eye, with incapability to turn the eye outwards; the other movements of the right eye and all those of the left are preserved.

There is slight but distinct facial paralysis of the left side.

March 3rd.—Patient's general condition is worse, his appetite is not so good; he feels cold and shivers occasionally. The temperature shows considerable variations; last night it was $102^{\circ}8'$, this morning it is $100^{\circ}6'$. The urine has again become very foetid and purulent.

The left arm has become very rigid, and is firmly applied to the side of the trunk and flexed at the elbow-joint, while the wrist is extended and the fingers slightly flexed on the palm; he is only just able to move this left upper extremity; the triceps tendon-reflex is increased on both sides; but there is no evidence of any loss of power in the right arm.

The condition of the cerebral symptoms remains unchanged, there is no evidence of the involvement of any more cranial nerves; the internal strabismus of the right eye and left facial paralysis persist. Hearing is equally acute on either side.

The conjunctiva of the right eye is now pale, but the corneal opacity exists. The right palpebral fissure is markedly smaller than the left.

March 11th.—Patient's general condition is worse; the temperature is more continuously above normal.

The condition of the cerebral symptoms remains the same as last week, except that the left facial paralysis is a little more marked.

While the anæsthesia over the course of distribution of the right 5th still exists, *the sense of taste over the anterior part of the tongue on either side is very acute.*

The upper limit of the anæsthesia, over the lower part of the body, is now about the same on either side, it extending up to a line drawn about 3 inches above the umbilicus.

The left arm remains in the same state; he can only just move it.

The effusion into the right knee-joint still exists.

For the last week he has spoken somewhat indistinctly, but there is no loss of memory for words.

March 18th.—He is much worse to-day. His temperature the day before yesterday was $105^{\circ}6'$, and this morning it is $101^{\circ}8'$; with this exception, it has never been below 102° F. for the last six days.

The incontinence of urine and faeces persists. The urine is exceedingly putrid and offensive.

The injection of the conjunctiva of the right eye has now come on again. The movement of the eyeballs is perfect in all directions, except that, through the paralysis of the external rectus of the right eye, he cannot look with that eye to the right.

The facial paralysis of the left side of the face is distinct; it involves the lower facial muscles, the upper facial muscles being quite free; he can close the eye on either side. There is no paralysis of either hypoglossal nerve.

The palpebral fissure of the right eye is still much less than the left; the pupils of the two sides cannot be compared, in consequence of the corneal opacity of the right eye.

March 22nd.—Patient gradually became weaker, and died last night, not having manifested any other symptoms.

Post-mortem.—This examination was made by Mr. A. H. Young, late Pathologist to the Manchester Infirmary.

Body greatly emaciated. Rigor mortis absent. Cutaneous hypostasis was well marked on the ventral aspect of the trunk and extremities (the body has been in the prone position since death, ice being applied to the spine).

A large bed sore existed over the sacrum and a smaller one over the right external malleolus. There was slight greenish discoloration of the anterior abdominal wall.

Spinal Cord.—On opening the spinal canal the whole of the nerves of the corda equina were seen to be greatly thickened and to possess almost a cartilaginous firmness. The dura mater was with difficulty separated from the posterior common ligament of the vertebral bodies, *i.e.* in the lower part of the canal.

Five inches above the tip of the conus medullaris, the spinal cord for a distance of $2\frac{1}{2}$ inches presented an area of great firmness, cutting and looking not unlike cartilage; at the periphery small opaque calcareous-looking deposits were observed.

Elsewhere the cord was somewhat softer than normal. In the cervical region there was marked dilatation of the central canal. No other definite naked-eye changes could be distinguished.

Brain.—The right side of the pons varolii was almost entirely replaced by a mass of new growth, like that in the spinal cord, of firm cartilaginous consistency. A small nodular outgrowth of this growth penetrated into the substance of the temporo-sphenoidal lobe. Brain-substance in other respects showed no naked-eye changes. The arachnoid membrane was slightly thickened and opaque, and there was considerable subarachnoid œdema.

The Gasserian ganglion on the right side was invaded by new growth, an extension of that of the pons.

Thorax.—Old pleuritic adhesions were found at each apex. Numerous subpleural masses (of various sizes) of new formation, flattened in shape, of a whitish colour. Lung-substance generally was normal.

Pericardium showed a single secondary nodule of small size.

Heart, 10 oz., dilated and hypertrophied.

Abdomen.—Alimentary canal not examined.

Liver, 4 lbs. 10 oz., congested.

Kidneys, right, 10 oz.; left, 7 oz. Substance of each organ presented numerous small abscesses. Pelvis dilated, and also full of pus.

Spleen, 8 oz., soft and pulpy.

Right knee and hip were opened; they showed hyperæmia of the synovial fringes and some increase of synovial fluid, but in other respects were normal.

If we briefly summarise the chief clinical and pathological features of the case, we notice: A man, of about 38 years of age, had suffered for 18 months with aching pains in the lower extremities, more marked in the left than the right, together with considerable weakness of the left leg. After this 18 months he first comes under our observation, complaining only of the above symptoms; we notice considerable wasting of the muscles of the left lower extremity, especially of the extensor muscle on the front of the left thigh; no loss of sensation and no bladder and rectum troubles are yet present. However, in the course of the next month he begins to get much worse; his legs become weaker, and he now, for the first time, notices great weakness of the right leg, which becomes the seat of very severe pains, similar to those observed in the left leg at a

previous date; about the same time he also has to strain to make water. In the course of the next month the weakness so much increases, that he becomes unable to walk, and has to take to his bed, and at the end of this second month he is not only not able to walk, but cannot raise himself from the lying to the sitting posture, and can only just move the toes and bend the feet at the ankle-joint. The lower extremities are quite flaccid and not at all rigid; the muscles are wasted, especially the extensor cruris of the left side. The muscles, however, show no reaction of degeneration.

At the beginning of the third month a patch of anæsthesia appears on the front and outer side of lower half of the left thigh, just above the knee, and another patch of anæsthetic skin is found over the costal arch on the right side in the right mammary line. In the course of the next six weeks the skin over the whole of the left lower extremity becomes anæsthetic up to the level of the umbilicus; in a month more the whole of the right lower extremity has become anæsthetic, and the anæsthetic part below has merged with the patch of anæsthesia over the right costal arch. During this time also the atrophy of the muscles of the legs has increased, and the catheter has had occasionally to be passed to relieve retention of urine.

Soon after the patch of anæsthesia appeared on the left thigh, *i.e.* after he has been under observation about three months, a series of cerebral symptoms begin to manifest themselves: he first notices pain, followed by anæsthesia, over the right eye, and the skin here becomes anæsthetic, and very soon this extends to the skin of the right cheek; ophthalmia also develops on the right side. The motor division of the 5th cranial nerve is also affected. By the end of the fourth month of observation complete retention of urine has ensued.

In the fifth month of observation there is evidence of effusion of fluid into the left knee-joint, without any signs of inflammatory action. In the same month the retention of urine gives place to incontinence, and the urine becomes purulent; at the same time slight pyrexia begins to develop.

In the fifth month, also, a painless ulcer appears on the inner aspect of the right half of the upper lip, and a little later, another on the inner aspect of the lower lip on the same side.

A month afterwards an ulcer appears on the skin of the lower eyelid near the inner canthus.

In the fifth month of observation the palpebral fissure of the right side is smaller than on the left side. Towards the end of the sixth month the left arm begins to become weak and rigid, and the triceps tendon-reflex of that side is increased. In the seventh month this increases, and paralysis of the external rectus of the right eye, and of the facial muscles of the left side, is to be noted.

The urine becomes more purulent and offensive, and the pyrexia greater. Bedsores appear on the sacrum, and bullæ are seen about the heels and malleoli. He gradually becomes weaker, and dies, after being under observation for about eight months, and having suffered from spinal symptoms for a little over two years.

At the autopsy is found a sarcoma, matting together the nerves of the corda equina; another sarcoma exists in the dorsal region, replacing the cord for a distance of two and a half inches, but the shape of the cord being here, nevertheless, still preserved; and a third tumour replaces the right half of the pons, and involves the 5th nerve and the Gasserian ganglion. Marked dilatation of the central canal of the spinal cord.

Secondary tumours in the lungs and pericardium. Suppurative nephritis.

Microscopical Examination and Commentary.—The various new growths—viz. that in the corda equina, spinal cord, pons varolii, and lungs—were all of the same nature, viz. spindle-cell sarcomata. Further, they appeared to be pure spindle-cell growths, no other kind of cell entering into their composition. We have already remarked upon the fact, so unusual in intraspinal tumours, that the growth in the dorsal part of the cord simply replaced the nervous elements of the cord, the shape of the cord being, nevertheless, still retained; so that no distinct outgrowth or tumour projection from the cord at the seat of the disease occurred, except the affection of the various nerves along which the new growth had extended. It may, in fact, be said that we had, for a distance of two and a half inches in the dorsal region, what was apparently at first sight a portion of the spinal cord, but in reality was that structure replaced

by sarcomatous elements. The growth extended along the cord, above and below, by growing along the outside of the blood-vessels, so that nearly every small arteriole at the periphery of the growth was surrounded by a mass of spindle cells, and its lumen was considerably narrowed from the pressure exerted by these from without. This mode of extension left traces of its previous existence in the older parts of the tumour, where the spindle cells were seen to be arranged in concentric groups, forming very small nests, the centre of which represented the narrowed, and in many parts completely obliterated, lumen of a blood-vessel.

At the extremities of the growth in the dorsal region, the nervous matter, which still existed between the masses of spindle cells arranged in groups round the blood-vessels, was very much degenerated, large accumulations of myelin being present at these parts; these myelin masses appeared to be gradually absorbed, and to be replaced by the sarcomatous elements. At some parts of this tumour in the dorsal region, and at the periphery of it, similar masses of myelin still existed to those which were found at the extremities of the growth, and it was the persistence of this myelin that gave the appearance described in the post-mortem report as resembling calcareous collections; there was, however, no calcareous matter.

The nerves issuing from the part of the cord which was replaced by the new growth, all suffered from an invasion into their substance of the sarcoma elements. The spindle cells had developed in the perineurium, surrounding and isolating the true nerve fibres, without apparently exerting very great pressure on those fibres, for even in the oldest parts of the growth the arrangement of the fasciculi of these nerve-roots was still distinct, but no axis cylinders could be recognised at any part of them; the absence of the latter, and the disappearance of the myelin, left a large number of small, more or less circular spaces, which had been previously occupied by the nerve fibres; the large number of these spaces in the sarcomatous infiltrated nerves, gave a hyaline appearance to the sections taken from these parts. The spindle cells of the tumour appeared to have simply replaced the perineurium, and

the true nervous elements, the axis cylinders and their myelin sheaths, had then become absorbed.

The appearance which these nerves presented on emerging from the affected portion of the spinal cord, was likewise again seen in the growth affecting the *corda equina*, only here it was seen on a larger scale, the growth having matted together the various nerves composing that structure. The spindle cells had here extended again between the separate nerves of the *corda equina* and between the various fasciculi of each nerve; but still the original arrangement of each nerve could be still recognised, for though the axis cylinders and their myelin sheaths had disappeared, the small spaces which they had occupied still persisted, surrounded on all sides by the spindle cells of the growth.

Those parts of the growth in the cord and in the *corda equina* which had developed most recently, were very rich in nuclei; but in the older parts these were far less numerous, and the growth had there taken on a uniform hyalin-fibroid appearance. With this exception, the tumours had not undergone any degeneration or further change with age.

There was nothing in the microscopic appearance of the growth in the spinal cord, or of that in the *corda equina*, to allow us to say positively which was the older of the two, and which consequently represented the primary one, but that in the pons was certainly of more recent development than either of the other two.

Although the pathological details collected after death do not allow us to solve this point, I think there can be very little doubt that the tumour in the *corda equina* was the primary growth, and the growth in the dorsal part of the cord was secondary to it; the pons tumour developing still later. It is highly probable that for the first eighteen months of his illness he was suffering from the growth in the *corda equina* alone, and it was only in the first month that he came under our observation that the tumour in the dorsal region began to develop, and that the complete paraplegia was brought about by the combined effect of the two tumours, and was not to be attributed to the tumour in the *corda equina* alone; that it was the development of this tumour in the dorsal region

which in great part accounted for the marked downward progress of the man when he was at the Cheadle Convalescent Hospital, and the rapid development of the complete paraplegia after his return to the Royal Infirmary. What speaks very strongly in favour of this view is, I think, the fact noted that soon after he became completely paraplegic, a patch of anæsthesia appears, not only over the left thigh, but also over the *costal margin* of the right side, a position which could not be accounted for by the tumour in the *corda equina*, but was probably due to the growth in the dorsal region, and possibly its extension along some of the sensory nerve roots from that portion of the spinal cord.

The spinal cord below the tumour in the dorsal region presented very well-marked and very advanced sclerosis of both lateral tracts, and also of Goll's and Burdach's column. The lateral sclerosis on either side was due to the interruption of the cord in the dorsal region by the tumour there; it was, in fact, a descending sclerosis.

The sclerosis of Goll's and Burdach's column on either side presented itself as a marked sclerosis, which extended on both sides from the posterior median fissure to the inner aspect of the posterior grey cornu, with no break of intervening healthy nerve tissue. This sclerosis we must regard as an ascending sclerosis from the effects of the growth in the *corda equina*. It is interesting as illustrating the fact that extensive lesions of the *corda equina* cause, not only a sclerosis of Goll's tract, but also of Burdach's as well. A considerable portion of Burdach's column, in the lower portions of the cord, being probably composed, either directly or indirectly, of fibres which proceed from the various nerves of the *corda equina*, and so consequently, when the latter is divided or interrupted by such a growth as we have in this case, sclerosis of that column results.

Above the tumour in the dorsal region there was well-marked evidence of ascending sclerosis, the column of Goll being very sclerotic in its whole vertical extent, and sclerosis could be traced as far as the posterior surface of the medulla. Immediately above the tumour, for a distance of about a quarter of an inch, there was not only a sclerosis of Goll's

tract, but also of Burdach's, exactly as we saw was the case below the tumour. We must consider this change in the posterior root zone as an ascending sclerosis; but it is not probable that it represents the effects of the growth in the corda equina before the tumour in the dorsal region began to develop, since probably the latter tumour grew, and practically divided the cord, before the one in the corda equina had completely cut off the nerves going to the lower extremities; at the same time we cannot forget that lesions of the great sciatic nerve alone have been followed by a sclerosis of Goll's column and of the posterior root zone. However, I should be much more inclined to attribute this sclerosis of the posterior root zone, which existed for a very short distance immediately above the tumour in the dorsal region, to the effects of that growth, which would cut off a large number of the fibres which would subsequently proceed upwards for a certain distance in Burdach's column: in support of this we may note the complete destruction of all nervous elements in both the spinal cord, and in the nerves which entered that structure opposite the seat of the new growth.

In the cervical region distinct sclerosis of the cerebellar tract also existed; this could be traced into the medulla, where it occupied a small tract in the restiform bodies. This is again to be regarded as an ascending degeneration.

From the position and extent of the cerebral tumour, we should have expected some evidence of descending change in the left lateral column; there was, however, no continuous tract of sclerosis, though some sections did show slight increase of the neuroglia in the lateral column of one side, and there was a small patch of sclerosis in the anterior pyramid of the medulla on one side.

The cavity existing in the cervical region of the cord, which in the post-mortem report is attributed to a dilatation of the central canal, was in reality to be so explained, as microscopic examination clearly proved. In the above region only one cavity existed, which occupied the median line, and was quite $\frac{3}{8}$ th inch in transverse measurement; it was flattened from before backwards, and so measured much less in that direction. It diminished in size from above downwards, being

distinctly smaller in the lower cervical region (from which Fig. 2 was taken) than just below the medulla oblongata (Fig. 1). In the upper dorsal region two canals existed, both of much smaller dimensions than the single one above: one of these occupied a central position, and represented the dilated central canal; the other occupied the position of the posterior grey cornu of the right side, having quite replaced that portion of grey substance. These two canals extended downwards about one inch, and were succeeded by a single canal occupying a central position, and evidently the original central canal of the cord, but now dilated, for a distance of about another inch, below which the canal again became double for a distance of half an inch, the two canals being again here situated as the two were higher up. Then below these two canals we had only a single one, occupying a central position as far as the tumour in the dorsal region. Below that new growth the central canal presented but slight increase above the normal dimensions, and was a single channel occupying its usual central position.

Consequently we had in the greater part of the cord an apparently very much dilated central canal; but in the upper dorsal region at two places two canals existed, between which two points there was only a single centrally situated channel. The wall of the canal in the cervical region (Figs. 1 and 2) was much thinner than in the dorsal region, where two canals existed (Fig. 4), although in the former place it enclosed a much larger channel than in the latter, where the lumen of either was very small. Further, the wall of the canal in cervical region was composed of firm, fibrous tissue, evidently of much older date and more fully formed than in the dorsal part of the cord, where each canal was surrounded by a fibro-nuclear growth, which was really very rich in nuclei. At the same time it could not be considered to represent a tumour mass, a new growth such as a glioma, but undoubtedly was an inflammatory tissue, and was probably to be regarded as the result of pressure exerted from within the canal. The lining membrane of the canal or canals was always well-defined, and often had a corrugated sinuous outline, or, as it has been described by some authors, of a shirt-frill character,

and we could hardly conceive how such a canal, with so thick walls and such an internal limiting membrane, could have originated by any decay in a previously existing tissue; far more probable appeared to be the explanation, that the cavity first existed with a thin wall, and that the latter thickened and became stronger to support the increased pressure.

The canal in the cervical region was so large, that though occupying a central position, it involved to a large extent the grey cornua, especially the posterior, which had practically disappeared (Fig. 1); the anterior cornua, on the other hand, were comparatively little implicated; but in parts even they were much compressed and diminished in size. The wall of these canals was composed of a fibro-nuclear tissue: in the cervical region, where the canal was large and single, the fibrous element predominated, and the wall was not so thick as elsewhere; in the parts where there were two canals, the nuclear element was in excess, and the wall was much thicker (Fig. 4), while the lumen of the canals was less than in the case of the single cavity in the cervical region. Towards its inner aspect, the wall of the cavity often presented a corrugated, folded appearance, just like the elastic coat of an artery. No endothelial lining existed.

The pathogenesis of such cavities as these in the spinal cord is still very uncertain. Although a really large amount of attention has been given to the study of the subject, hardly any two authors will be found to agree as to the exact sequence of events which has led to their formation, and it is quite possible, in fact, from the recorded cases *probable, that the same explanation will not suffice for all instances of the morbid appearance.*

It is well that we should start with a clear idea of what we mean by the term syringomyelia, and what by hydromyelia. Simon, Leyden, and Ziegler separate congenital enlargements of the central canal under the term hydromyelia, from those cases where we get cavities formed from pathological causes subsequent to birth, and to which the term syringomyelia is applied. To the latter group would belong our present instance.

It is noteworthy that this affection is most commonly either

limited to the cervical or dorsal parts of the cord, or is most advanced there; also that the cavities which are formed are most frequently situated either in the posterior white columns, the posterior grey commissure, or in the posterior cornua, very rarely do they appear in the anterior portions of the cord, either in the grey or white substance.

Leyden¹ regards syringomyelia really as a consequence of congenital hydromyelia, attributing it to an interference with the closing the central canal, which is at first so large in the embryo. The position in which these cavities are found, chiefly in the posterior parts of the cord, would be held to support this view.

Westphal² and Simon³ both attributed it to the development of a new growth, a tumour (and not inflammatory tissue) in the spinal cord with a subsequent degeneration of the centre of this, and the formation of a cavity or canal. According to their views, the cavity has nothing directly to do with the original central canal of the cord. Westphal, however, in a more recent paper,⁴ shows that his former view must to a certain extent be given up, and that a certain relation of such cavities to the central canal must be acknowledged. And he says, the changes found in the cases most recently observed by him were doubtless congenital, and that we must assume that disturbances of development of the central canal ensued with cavity formation, accompanied by a tendency to the development of fibrous new growth. He adds, however, that "in other cases there seems to be a tendency to the development of another form of new growth (glioma, myxoma)."

Schultze⁵ likewise holds the view, that these cavities owe their origin to a decay of proliferated neuroglia tissue, which may take its origin at various parts of the spinal cord, or in the medulla oblongata.

Hallopeau⁶ regarded a central myelitis as the primary

¹ 'Klinik der Rückenmarkskrankheiten,' ii. p. 447; and Virch. 'Archiv,' vol. 68. Compare also Taylor, F., 'Trans. Path. Society,' 1884, vol. xxxv. p. 36.

² 'Archiv f. Psychiatric,' vol. 1, 1874, pp. 30 and 109. ³ Ibid.

⁴ 'BRAIN,' July 1883, pp. 149 and 165.

⁵ Virch. 'Archiv,' vol. 87.

⁶ "Contribution à l'étude de la sclérose diffuse périependymaire," &c., 'Gaz. Méd.,' 1870, 30, 32, 34, 35.

lesion; a diffuse sclerosis ensuing round the central canal, and in consequence of degenerative changes which ensued in this inflammatory tissue, cavities were formed.

Langhans¹ attributes the cavities to a dilatation of the central canal of the cord, and the formation of diverticula from it, brought about by blood stasis, which stasis may be occasioned in various ways; in three of his four cases it was effected by tumours in the posterior fossa of the skull.

My own case I am inclined to attribute to dilatation of the central canal, and the formation of diverticula from that, but I am less inclined to explain that dilatation by a blood stasis occasioned by the tumour in the pons, than by an obstruction to the circulation of the cerebro-spinal fluid, and to a damming up of that fluid in the central canal of the cord, whereby great pressure was exerted, diverticula formed, and as a consequence of that pressure, a sclerosis ensued round the canals so arising, and that consequently the sclerosis was not a primary element in the case.

The most recent writer on the subject is Dr. Ed. Krauss,² who ably reports a very interesting case. He explains the syringomyelia which existed, as a consequence of proliferation of the neuroglia tissue, which first formed, as it were, a kind of glioma, and then the central part of this undergoing decay, cavitation resulted. After carefully considering the previous history, and the pathological appearances found after death in Dr. Krauss's case, I am much more inclined to think that the primary lesion there was a hæmorrhage into the upper part of the spinal cord, and possibly medulla, with a secondary descending sclerosis of the lateral column of the one side, together with the formation of a cavity, the greater part of which was due to a dilatation of the central canal of the cord, but that part of the cavity above might be due to changes ensuing in the hæmorrhagic focus itself.

Dr. Krauss believes that there had previously existed a syringomyelia which had caused no symptoms until those ensued quite suddenly (the patient waking up one morning

¹ "Ueber Höhlenbildung im Rückenmark als Folge von Blutstauung," Virch. 'Archiv,' vol. 85, p. 1.

² "Ueber einen Fall von Syringomyelia," Virch. 'Archiv,' August 1885, vol. 101, p. 304.

and finding the right leg and arm paralysed), and he is evidently of the opinion that the most probable explanation of the sudden onset of those symptoms was a hæmorrhage, which, as he remarks, is no seldom occurrence in the case of a glioma. For my own part, I can see no reason or necessity to assume, that before the hæmorrhage ensued such a proliferation of the neuroglia did exist, nor that there was before the onset of the symptoms, any syringomyelia. A far more rational view appears to me to be, that hæmorrhage into the spinal cord (rare as such lesion is) was the primary change.

As regards my own case, I think few could study the sections from which the figures were taken without coming to the conclusion, that most probably the sequence of events had been increased pressure within the central canal, dilatation of this (Figs. 1 and 2), formation of a diverticulum (Fig. 3) or outgrowth from the same, and eventually the establishment of two distinct channels (Fig. 4) with very thick walls.

From cases recorded, it is evident that syringomyelia may reach a very advanced stage without giving rise to any definite symptoms during life. In others it occasions marked atrophy of various groups of muscles, with or without sensory disturbances. It is doubtful whether, in our own case, any of the symptoms could be referred to the syringomyelia and to the sclerosis round the canals which existed. The grey matter was very extensively involved, as Figures 1 and 2 show; in some parts not only was the posterior (Figs. 1, 2 and 3), but also the anterior cornu of one or other side considerably affected (Fig. 3). It is hence possible that such a lesion might have been really the cause of the deficient chest movement, which was noted in the latter part of our patient's existence. It is certain that the position of the tumour in the dorsal region, and the involvement of the issuing spinal nerve-roots, would not account for this paralysis of the intercostals.

An interesting point also in our case was the existence in the anterior pyramids of the medulla, close to the anterior surface (Fig. 5 *a*), of a distinct and well-marked nucleus of grey matter, which was so large as to be quite easily visible to the naked eye. This nucleus commenced immediately above the

decussation of the pyramids, and ran upwards to close upon the lower border of the pons, a vertical distance of nearly $\frac{3}{4}$ inch. Quite at its upper part this nucleus divided into two, one occupying the position the whole did lower down, viz. the anterior surface of the medulla, while the other and smaller half was situated at the side of the anterior median fissure, rather more posterior and internal than the above-mentioned half.

At its point of greatest size, which was at its lower part, this nucleus presented upwards of two hundred ganglia cells; these diminished in number towards the upper limit of the nucleus.

No nerves were seen coming off from the anterior surface of this nucleus, but from its deeper part, filaments of nerves ran into and were lost, in the deeper parts of the medulla. No connection could be traced between this nucleus and any of the other nuclei in the medulla (Fig. 5 *a*).

Another well-marked nucleus of grey matter existed at the posterior part of anterior pyramids immediately to the inner side of the hilum of the olivary body (Fig. 5 *b*). I specially mention the existence of these two nuclei, since the majority of our English anatomical text-books quite ignore their existence, and I possibly may hence be forgiven for having at first considered them to be but another item in making our patient a pathological curiosity.

Both of these nuclei are, however, recognised in the majority of the anatomical works of continental authors; they are, in fact, the nuclei arciformes of Henle,¹ or the anterior and posterior pyramidal nuclei of Kölliker, and the small pyramidal nuclei of Stilling.²

In our present case they were both exceedingly well developed.

The cerebral symptoms call for special mention in reference

¹ 'Handbuch der systematischen Anatomie des Menschen,' von Dr. J. Henle, 1868, Nervenlehre, p. 194.

² See also 'Allgemeine und microscopische Anatomie,' von W. Krause, III Auflage, 1876, I. Bd., page 413 and Fig. 242; also 'Zehn Vorlesungen über den Bau der Nervösen Centralorgane,' von Dr. Ludwig Edinger, 1885, page 121, Fig. 107. Also Ross 'On Diseases of the Nervous System,' 2nd edit., vol. i. p. 807: "The nucleus of the pyramid (internal parolivary body)."

to one point. The trophic lesions in the area of distribution of the right trigeminus were highly interesting, but what I would specially call attention to is, that though the post-mortem proved how complete had been the implication of the 5th nerve with the Gasserian ganglion of the right side, yet there had not been the slightest affection of taste on the corresponding side of the anterior two-thirds of the tongue. I repeatedly tested for this, since the case was under observation at the time when I became acquainted with Erb's views on the course, which the gustatory fibres supplying that part of the tongue took to the brain.¹

Whatever may be the course of the gustatory fibres of the chorda tympani in the majority of cases, it is certain that in our case they did not pass with the 5th nerve on the right side. Although many cases are recorded of loss of taste over the anterior two-thirds of the tongue from disease of the 5th nerve, others have existed, and been put on record, where complete paralysis of that nerve left taste unaffected.

In conclusion, I would subjoin a few further references to the subject of syringomyelia :

NONAT: "Recherches sur le développement accidentel d'un canal rempli de sérosité dans le centre de la moëlle épinière."—'Archives générales,' 1838, i.

JOLYET: "Sur un cas d'anomalie du canal central de la moëlle épinière."—'Gaz. Méd. de Paris,' 1867.

VIRCHOW: "Die Betheiligung des Rückenmarks an der Spina bifida und der Hydromyelia."—Virch. 'Archiv,' xxvii. 1863, p. 575.

LANCEREAUX: "Un cas d'hypertrophie de l'épendyme spinal avec oblitération du canal central de la moëlle."—'Mém. de la Soc. de Biol.' Paris, 1862.

SCHÜPPEL: "Ueber Hydromyelus."—'Archiv der Heilkunde,' vi. p. 289.

GRIMM: Virchow's 'Archiv,' 1869, vol. 48, p. 445.

NAUNYN and EICHHORST: "Ueber die Regeneration und Veränderungen im Rückenmark."—'Archiv f. exper. Pathol. und Pharmacol.,' ii. 1874, p. 225.

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FRIEDREICH: Virchow's 'Archiv,' vols. 26 and 27.

¹ "Ueber den Weg der geschmackvermittelnden Chordafasern zum Gehirn," 'Neurogl. Centralb.,' vol. i., 1882.

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EXPLANATION OF THE PLATES.

Fig. 1. From the extreme upper part of the spinal cord.

- a.* Extremely dilated central canal with comparatively thin fibrous wall.
- b.* Sclerosis of Goll's columns.

Fig. 2. From lower cervical region.

- a.* Dilated central canal, here smaller and with a thicker fibrous wall.
- b.* Sclerosis of Goll's tracts.

Fig. 3. Lower cervical region.

- a.* Formation of a diverticulum (*c*) from the dilated central canal.

Fig. 4. From dorsal region.

- a.* Two cavities (*a* and *c*) with thick fibro-nuclear walls.

Fig. 5. From the medulla.

- a.* Anterior pyramidal nucleus.
- b.* Posterior pyramidal nucleus.

ON A CASE ILLUSTRATING THE CORTICAL NATURE OF EPILEPSY AND ITS RELATIONSHIP TO JACKSONIAN CONVULSIONS.

BY D. NOËL-PATON, M.D., B.SC.,

Biological Fellow of the University of Edinburgh.

SINCE Bastian first argued for the existence of cortical "perceptive centres," and since the researches of Ferrier have demonstrated that centres connected not only with the motor functions, but also with the perception of special sense-impressions occupy definite areas in the cortex cerebri, much has been done, both experimentally and clinically, to prove that all forms of epilepsy are cortical in their nature.

Among the many workers in this field of inquiry, Hughlings-Jackson stands out pre-eminent in having demonstrated by clinical and pathological research the connection of certain definite forms of epileptic attack, with coarse lesions of the motor areas of the cortex. But, though experiment renders it extremely probable that similar irritative lesions in the sensory areas of the cortex may also cause epileptic attacks, the clinical evidence on this point is still very deficient.

The following case is of considerable interest in this respect, since, in connection with a well-localised external injury in relationship to both motor and sensory cortical areas, two perfectly distinct classes of fits occurred—the one resembling in all essential features the motor seizures of Hughlings-Jackson, and the other corresponding to the ordinary epileptic attack.

For leave to publish this case I am indebted to Dr. Brakenridge, in whose ward it occurred.

William Heslop, aged 23 years, and unmarried, a brass-finisher by trade, but recently working as a labourer, was

admitted to Ward 32 of the Edinburgh Royal Infirmary on Oct. 11th, 1883, complaining of "fits," which had troubled him for four or five years.

HISTORY.

1. *Family History*.—The patient is the eldest of a family of seven, four of whom are dead. All four died in infancy; in the case of one the cause was "water on the head," in the other cases the cause of death is unknown. The other surviving members of the family, a brother and sister, are strong and perfectly healthy. His father died in Morningside Asylum, suffering from general paralysis of the insane. He had been a very hard drinker. One of his father's brothers died of delirium tremens, but his father's other brothers and sisters are said to be free from disease. His cousins are all healthy. His mother is healthy, and there is no neurotic history in connection with her family.

2. *Personal History*.—The patient has always had a comfortable home and good food, and his work has never been excessive. He generally on Saturday night indulges freely in beer, but he is not a heavy drinker.

3. *Past illnesses and accidents*.—As a child he had measles and scarlet fever; but since then he never had any illness. There is no history of syphilis, or of convulsions during teething.

At seven years of age he fell over a stair, three stories high, and sustained a very severe fracture of the frontal bone, for which he was trephined by Dr. Gillespie. After the operation he made a rapid and complete recovery.

Five years ago, when working on board a steamer at Glasgow, an iron bucket, weighing about half a hundred-weight, fell upon the right side of his head, inflicting a compound fracture of the parietal bone. He was stunned for a moment, but almost immediately regained consciousness. Before he could be got to the Infirmary he was again unconscious, and remained so for several days. He was under the care of Dr. McEwan, and I have been able to ascertain that the treatment was expectant, and that no head-symptoms developed. The patient himself states that a small piece of

bone was removed. He was not discharged from the Infirmary for eighteen months, during which time the wound on the side of the head remained unhealed. Three weeks after his dismissal his *present illness* commenced.

4. *Present illness*.—Walking along the street in Glasgow, he suddenly fell down in an unconscious condition; and upon recovering, he suffered for some time from severe headache. Since then he has been constantly liable to fits, which come on at irregular intervals, sometimes when he is awake, sometimes when asleep. If he is seized during sleep, the fit is generally much more severe.

Two kinds of attack are described :—

In the first, consciousness is almost at once and completely lost; while in the second it remains undisturbed, either throughout the whole fit, or for a considerable time after its commencement.

In connection with seizures of the first type, all the patient can tell is that they are usually preceded by a very distinct and somewhat complex aura. He first experiences a sensation of heat in the left side of his face, and almost simultaneously he sees a ball of fire before the left eye, while at the same time he hears a singing sound, but whether in one or both ears he is unable to say. His mother describes these fits as very violent, and from her description they appear to resemble very closely in all particulars ordinary epileptic seizures.

Of the second type of convulsion the patient gives a very clear account. The fit commences with a twitching of the whole upper lip, or of one side of it, sometimes the right, sometimes the left. Ultimately, however, the left side is most markedly affected, and the mouth and cheek are drawn over to the left side and twitched violently. The muscles concerned in flexion and extension of the left elbow-joint are next involved, first becoming rigid, with the arm fixed in a position of semi-flexion, and then passing into a state of clonic spasm. The next step is the flexion of the hand on the forearm, and then the shoulder-muscles are affected, first with a tonic and subsequently with a clonic spasm, while the head is twisted over to the left side, and the whole body is bent to the left. Consciousness is generally lost at this stage of the

attack, but the motor spasm may first extend over to affect the right arm. As soon as the spasm reaches the right side, consciousness is invariably lost, and it is impossible to trace the further progress of the fit. He states that upon recovering consciousness, he finds the left arm still jerking. After the fit he suffers from severe frontal headache, equally intense upon both sides and throbbing in character.

The convulsion does not always go so far as above described; frequently it never passes beyond the arm, and consciousness is then retained throughout.

STATE ON ADMISSION.

Weight, 8st. 9lb.; Height, 5ft. 5in. He is a well-developed, fairly strongly built young man, of fair complexion, and with a large brachycephalic head. The eyes are somewhat deeply set, the pupils large and equal, the iris is of a lightish-grey colour. The conjunctivæ are clean. The features are small and well-formed, and the expression is intelligent.

The left side of the forehead is more prominent than the right, but the supra-orbital ridge is, at its outer extremity, obliterated. At a point $2\frac{1}{2}$ inches from the middle line, and $1\frac{1}{4}$ inches above the eyebrow, there is a well-marked rounded protuberance; and running upwards and inwards from this is a ridge 2 inches long. Under this ridge is a depression $1\frac{3}{4}$ inches long by 1 inch broad at its widest part. It is ovoid in form—the large end of the ovoid being directed downwards and outwards. Two inches above the root of the nose is a crescentic scar, with its concavity directed upwards, and this terminates at its left extremity in a triangular somewhat depressed cicatrix. Over the whole of the ovoid depression very marked pulsation may be observed, and when the patient stoops, not only is this pulsation increased, but a well-marked bulging becomes visible. A bridge of bone passes horizontally across the middle of the ovoid, but above and below this a depression, which is soft and yielding to the touch, may be felt. The median triangular patch also pulsates and bulges when the patient stoops. The crescentic cicatrix covers a depression, having its deepest part at its right extremity. Uniting the right angle of the triangle with the

upper and inner end of the ovoid, a narrow linear depression may be felt.

On the right side of the head, $4\frac{1}{2}$ inches above the tip of the mastoid process, is a cicatrix, uncovered by hair and partly adherent to the subjacent parietal bone. It covers a linear depression about $\frac{1}{4}$ inch broad, which extends, from a point $\frac{1}{2}$ inch behind a perpendicular drawn through the mastoid process, upwards and forwards for 2 inches to a point 4 inches above the tragus of the external ear. The depression is deepest and broadest where a vertical line from the external auditory meatus intersects the scar.

My friend Mr. Hare kindly examined the position of this scar for me, and from a long series of observations—the results of which he has since published,¹—he believes that this depression must be entirely behind the fissure of Rolando, and that it must pass over the lower part of the ascending parietal convolution, and run downwards and backwards over the posterior part of the fissure of Sylvius.

NERVOUS SYSTEM.

1. *Sensations*.—He suffers from headache and dimness of vision for some time after his fits.

2. *Sensibility*.—1. Common sensibility, thermal sensibility, &c., are unaltered. 2. *Special Senses*.—Pupils are equal, widely dilated, and respond only slightly, but equally to light and to accommodation. The field of vision and visual acuteness are unaltered.

Ophthalmoscopic examination.—Both eyes are slightly myopic—the media are clear; in both there is a well-marked deep cupping of the disc. The laminae cribrosae are well marked. In the left eye are marked signs of old neuritis, probably connected with injury to the eye from his first accident.

Taste, hearing, and smell are normal. The muscular sense is also normal.

3. *Motor Functions*.—The organic reflexes are normal. During the fit the patient does not lose control over the bladder and rectum. The tendon-reflexes are normal, and the cutaneous reflexes are all fairly marked. Voluntary movements are

¹ 'Journal of Anatomy and Physiology,' vol. xviii. p. 174.

in all respects normal. Co-ordination is perfect; and the electrical irritability is unaffected.

4. *Cerebral and Mental Functions*.—The patient sleeps well. His mental faculties are well developed; altogether he is a most intelligent, clear-headed fellow, fond of reading and of various games.

All the other systems are normal.

DIAGNOSIS.

The seizures first described correspond in all essential particulars to Hughlings-Jackson's classical description of that form of epilepsy named after him; and that they are really of this nature is rendered certain by their occurrence in connection with a well-defined traumatic lesion in the region of the cortical centres related to the muscles first implicated in the attacks.

Unfortunately, after his admission to the Infirmary, the patient remained free from these more purely motor seizures, so no opportunity was afforded of studying their onset, and of thus definitely localising their starting-point.

The patient's own statement, confirmed by that of his mother clearly shows the lips were first implicated. But this is not sufficiently definite. For it must be remembered that the labial centres occupy no small part of the motor area of the cortex.

Mr. Hare's careful measurements, however, which were found post-mortem to have yielded extremely accurate results, indicated that the injury did not extend in front of the fissure of Rolando; that it implicated the lower part of the ascending parietal convolution, part of the angular gyrus and part of the superior temporal convolution; that, in fact, the only motor area involved was No. 11, that centre connected with retraction of the angles of the mouth.

In all probability the clinical study of this point would not have yielded very accurate results, since the close physiological relationship of all the labial centres, and the consequent almost simultaneous implication of these, would have rendered it impossible to have detected in which the nerve explosion really commenced.

The characters of such Jacksonian seizures are now so generally known to the profession that it is needless here to enter more fully into the various phases of the attacks.

It is of some interest, however, to compare the march of spasm on the body with its course over the cortex cerebri. Centre 11 being first affected; centres 7, 8, 9, and 10, are next implicated; while 6, the centre for the bicipital movements, and *a*, *b*, and *c*, for the movements of the hand and wrist, are subsequently involved. The more remote centres, 12, 5, 4, 3, 2 and 1, are last implicated, just before or just after the spasm passes to the other side of the body.

But besides these motor attacks, our case presents for our consideration seizures having different and graver manifestations; and the question which we must now consider is whether these are due to an idiopathic epilepsy; or whether, on the other hand, they owe their origin to the same injury which we have been led to consider the cause of the minor seizures.

The evidence in favour of true epilepsy seems at first sight very strong. We have the patient's family history. His father died insane, and an uncle suffered from delirium tremens.

But that it is not a true idiopathic epilepsy—that it is not what would be called by Nothnagel a primary epilepsy—that is, an epilepsy due purely to inherent and inherited causes, is I think manifest from the very definite connection of these fits with the head-injury; though of course it is extremely probable that our patient inherited from his progenitors brain-cells so unstable as to render him peculiarly susceptible to any exciting cause of epilepsy.

But granting that this is a secondary epilepsy dependent upon the head-injury, the question now arises—do these more typical fits owe their origin to the same physical condition with which we have connected the Jacksonian seizures; or must their cause be sought elsewhere?

This blow on the head might have induced such seizures in different ways.

Firstly, we might have here a clinical example of one cause of epilepsy which Westphal has demonstrated in guinea-pigs.¹

¹ 'Berliner Klinische Wochenschrift,' 1871, No. 31.

He found that if the head of the animal be struck lightly, a convulsion may be induced. This rapidly passes off, and leaves the animal in an apparently perfectly normal state of health for a period of some weeks, when an epileptic condition appears, fits occurring either spontaneously, or in connection with an epileptogenous zone. Post-mortem examination of these guinea-pigs reveals numerous small hæmorrhages among the fibres of the white substance of the spinal cord and upper part of the medulla.

The bearing of these experiments upon the case before us is so evident as to need no further remark from me. Unfortunately, the clinical evidence of the occurrence of such fits is small, unless in connection with embolism.

Secondly, Brown-Séquard has now most fully and satisfactorily demonstrated the possibility of inducing in guinea-pigs epileptic attacks, and an epileptic state lasting for a considerable length of time, by section or injury of peripheral nerves, either sensory or mixed. He found that, soon after section of the nerve—the sciatic was generally used—over the same side of the head and face an area of increased superficial reflexes appeared. This he called an epileptogenous zone. These reflexes gradually increased till a complete local spasm might be induced, and after some few days the spasm affected the whole of one side of the body. In the course of a month or six weeks, instead of unilateral spasm a well-marked epileptic fit resulted from irritation of this area.

Cases in man of this form of secondary epilepsy have occurred, though, as a rule, without the formation of epileptogenous zones, and it is a matter of no little interest to note that such cases are usually connected with an injury either to the sciatic or to the fifth cranial nerve. To quote Nothnagel's words:—"The lesion generally consists in external traumatic agencies, acting upon the trunk, branches, or cutaneous distribution of these nerves, less often on pressure due to tumours." In our case the area of distribution of the fifth cranial nerve is thus injured.

Against the supposition that our case is of this nature must be set the fact that the aura, when it occurs, is of the nature of some peculiar sensation in the area of the affected nerve. Here, however, we have a distinct aura in quite another region.

I believe that in our case we have to deal, not with an idiopathic epilepsy, but with an unusual form of secondary epilepsy, having its starting-point in the special sense-areas of the cortex.

I have been led to this conclusion firstly, by the localisation of the external wound in relation to the cerebral convolutions ; and secondly, by a study of the aura epileptica.

I have already stated that Mr. Hare, from these measurements, is of the opinion, that the fracture must have passed over the lower part of the ascending parietal lobe, and extended across the posterior part of the Sylvian fissure—that it passes over area 11, over part of 13, and part of 14. Now, as is well known, area 13 has been proved by Ferrier to be the visual centre, while 14 has been similarly demonstrated to be the auditory centre ; so that, in case of any meningeal thickening or other lesion in relation to the inner surface of the fracture, we must conclude that centres 13 and 14 are affected in the same manner as 11—the motor centre which we have seen participating in the commencement of the Jacksonian attack.

Secondly, the auræ, as described by the patient, are—first, a feeling of warmth over the left cheek ; secondly, a ball of fire before the left eye ; and thirdly, a singing in one or both ears.

Now these special sense-auræ are just what, from Ferrier's researches, we should expect in the case of an explosion of nerve energy in the ganglion cells of the visual and auditory centres, and that these cells are in all probability in an unstable condition we have already shown. The meaning of the thermal sensation is more difficult to explain, from the fact that centres connected with such sensation have not yet been localised.

I think then that we are justified in concluding that these, as well as the more purely motor fits, owe their origin to some gross lesion in connection with the fracture.

It is not difficult to understand how one exciting cause may act upon the cells of the sensory area ; while another, probably starting from a different source, will more distinctly affect the motor areas.

That a sudden discharge of nerve energy, occurring in con-

nection with the sensory areas of the brain, should cause a more generalised convulsion than a similar discharge in a group of motor cells, is obvious; firstly, from the very intimate connection of each sensory area with many different motor centres, as is well shown by the very complex series of movements which supervene upon the irritation of such a sensory area; and, secondly, from the fact that nervous discharges, starting from the sensory cells, pass in a great measure towards the motor cells; that, in short, while the natural course of a discharge from a motor cell is outwards to the periphery, the course of a sensory discharge is towards motor cells, upon the action of which the natural manifestation of the sensory impression depends. Thus, once given the initial sensory disturbance, it is pretty obvious that the fit must become general, and cannot remain localised, as was the case in many of the motor attacks.

This diagnosis, arrived at during the life of the patient, was confirmed post-mortem.

Illness prevented my studying the progress of the case; but I have ascertained that after his first month in hospital he remained entirely free from fits; that he grew stupid, lethargic and heavy; that his sight became impaired, and that his general health steadily deteriorated. He went home for a short time in January 1844, but was readmitted almost immediately, and died on January 24th.

AUTOPSY.

On January 25th, Dr. Byrom Bramwell made a post-mortem examination, the report of which I append.

External Appearances.—Length 55", circumference at shoulders 37"; emaciated, pale; pupils equal and contracted; rigidity moderate; lividity slight; putrefaction commencing. A cicatrix, $\frac{1}{2}$ " long, above left eyebrow (its middle); another in the middle line, $1\frac{1}{2}$ " above the root of the nose. A well-marked depression at the seat of the first-mentioned cicatrix. Another depression, 3" above and 2" behind the right ear (external auditory meatus).

Brain.—Fracture of the frontal bone extended from the depression above the eyebrow up to middle of forehead; bone

absent at seat of depression, a hole the size of a sixpence bridged over by membrane. The dura mater and arachnoid are firmly adherent to the perforation in the frontal bone. At the seat of the depression behind the right ear was an old fracture. A needle could be passed through the narrow fissure at the bottom of the fracture, which was bridged over by membrane. The dura mater is adherent to the inner surface of the bone at the point of the external fracture; but no lesions of the arachnoid or convolutions here.

The tip of the left frontal lobe was destroyed by a cyst, and was fixed to the inner table of the skull at the seat of fracture in the frontal bone by the adhesion mentioned above. The cyst was about the size of a walnut. Surface of the brain otherwise quite normal. Weight 3 lbs.

Right optic disc and retina normal.

The pericardial sac contained 1 oz. of blood-stained serum; one or two punctiform hæmorrhages in posterior surface of the pericardium. The heart weighed $7\frac{1}{2}$ oz., and was healthy. Both pleural sacs dry. Right lung weighed 1 lb. $7\frac{1}{2}$ oz., very much congested, especially at lower lobe. Some œdema of upper lobe. Left lung weighed $14\frac{1}{2}$ oz., much congested. Spleen weighed 4 oz., congested. Liver weighed 2 lbs. $3\frac{1}{2}$ oz., congested, fatty and decomposing. Kidneys; right weighed 5 oz., congested; left, $3\frac{1}{2}$ oz., in similar condition. Stomach, patches of congestion. Intestine normal.

Dr. Clemon, Dr. Brackenridge's then resident physician, was able, post-mortem, to confirm Dr. Hare's measurements in regard to the locality of the fracture in relationship to the Sylvian fissure and the neighbouring convolutions.

I must add to this report the fact, that at the seat of the fracture of the right parietal, the inner table of the skull showed marked signs of inflammatory action, while the adherent dura mater was considerably thickened.

COMMENTARY.

Satisfactory as is the post-mortem in regard to the localisation of the lesion, it demonstrates the existence of a disturbing element unrecognised during life. The presence of a large cyst in the frontal lobe of the left side, connected with the

fracture sustained during early childhood, at once introduces the question of how far this was connected with the development of the epileptic seizures. I believe we may safely exclude this possible factor. For a careful analysis of the numerous recorded cases of lesions of the frontal lobes¹ shows that their connection with epileptic attacks of such a very distinct character is unknown, though undoubtedly epileptic fits may be caused by such lesions, just as they may be caused by lesions in any part of the nervous system.

Again, the development of these fits in such direct relationship to the later injury points very strongly to its causal connection with them, especially if we take into consideration the nature of the aura.

Perhaps the most interesting point demonstrated by the post-mortem is the absence of any marked change in the cortex cerebri at the seat of the fracture. It is greatly to be regretted that the preliminary treatment of the brain with too strong alcohol rendered it friable, and unsuited for microscopic examination. The inner surface of the parietal bone showed well-marked signs of inflammatory action, while the dura mater was distinctly thickened.

Other cases of epileptic attacks connected, as demonstrated during life by the aura, and post-mortem by the position of the lesion, with irritation of the special sense areas are on record;² but I have failed to find any case in which such a lesion produced two such totally different forms of seizure.

Such a case, connecting as it does the simple Jacksonian attacks with the more complicated manifestations of true epilepsy, is of very great interest and importance.

All evidence, clinical and experimental, points to the following conclusions:—1st, that the starting-point of all epilepsies is in the cortex; 2nd, that pure nervous explosion, without the intervention of vaso-motor changes, is sufficient to account for all the phenomena of the attack.

The second point has been fully treated by Gowers in his work on Epilepsy.

It is in connection with the former point that such cases as

¹ M. Allen Starr, M.A., M.B., 'Journal of Medical Science,' April, 1884.

² Starr, *loc. cit.*

ours are of special interest, affording, as they do, strong evidence in opposition to the medullary theory, still so ably supported by Nothnagel and others.

At first sight the experimental evidence upon which this theory is built appears extremely strong; but when viewed in the light of more recent research, it loses much of this apparent strength.

Undoubtedly the researches of Nothnagel have demonstrated beyond dispute the existence *in the rabbit* of an area in the pons Varolii and medulla oblongata, irritation of which causes general convulsions¹; and at the same time have shown that the action of this centre is independent of the higher part of the brain—that convulsions do occur when the brain above has been removed. But Rosenbach, who has recently studied this subject,² contends that in dogs such fits do not manifest the characteristics of the true epileptic attack. And it must also be remembered that from experiments on the very undifferentiated brain of the rabbit it is hardly justifiable to draw conclusions in regard to the vastly more complicated and differentiated human brain.

Again, Nothnagel has not availed himself of the most valuable clinical method of research which we owe to Hughlings-Jackson—the careful study of the aura epileptica, the earliest symptom of the attack.

All evidence goes to prove that the perceptive centres are situated high up in the brain. Now, the aura epileptica is in the vast majority of cases of the nature of a perception of a sensation, and is not merely a reflex expression of such a sensation—such as would be produced by a nervous discharge commencing in the lower centres. Further, according to Gowers, in one-fifth of all cases in which an aura occurred this was of the nature of a special sense-warning, and the researches of Ferrier and all pathological evidence go to prove definitely that the centres connected with such perceptions are situated in a specific area of the cortex. When, on the other hand, the aura is of the nature of some localised muscular twitching or contraction, no doubt in regard to the cortical position of its starting-point can be entertained.

¹ Virchow's 'Arch.,' vol. xliv. p. 1.

² Ibid.

Those cases in which no aura occurs cannot be cited as supporting the medullary theory, for we must remember that the absence of this warning is in all probability due to an inherent general weakness of the nerve-cells, allowing the nerve explosion to pass so rapidly from one centre to another that consciousness is lost before time is allowed for cognition of the initial perception.

Again, each attack deteriorates not only the group of cells first implicated, but also all secondarily involved, so that in time these will offer so little resistance to the spread of the attack, that the fit will very rapidly become sufficiently general to abolish consciousness.

By some the so common form of aura apparently connected with the pneumogastric nerve is considered as indicative of the primary affection of the medulla; but, as we have already pointed out, even these so-called organic auræ are of the nature of true perceptions, and therefore indicate the implication of the higher parts of the brain.

So much for the clinical evidence in regard to the nature of the epileptic attack, which certainly seems to render it highly probable that the starting-point of the fit is in the cortex cerebri. And this evidence is fully confirmed by the recent experimental research of Rosenbach.¹

This research is of such importance, and appears to have received so little attention in this country, that I do not hesitate to give a brief *résumé* of the main results obtained by this observer. His experiments were conducted on dogs, and yielded the following result—

A. Motor Areas.

1. Electrical irritation of a cortical motor area causes a well-marked epileptiform convulsion in the muscles corresponding to this area; and this convulsion passes by a definite route to other groups of muscles—the route corresponding to the proximity of the various motor areas of the brain.

2. On removing the electrodes, the attack passes off in the usual manner of epileptic attacks, the tonic spasm becoming clonic, and ending in paralysis of the muscles affected.

¹ Virchow's 'Archiv,' Bd. 97 s. 339.

3. It is difficult to regulate the current so as to confine the attack to one side of the body. When the median line is passed, the hind limb is the part first affected. When the centre for the hind limb is irritated, the attack frequently at once passes over to the opposite side.

4. At the height of the attack consciousness is lost.

5. After the fit one of two conditions may remain—

a. Post-epileptic paralysis.

β. Post-epileptic psychical irritability.

6. For a few minutes after the cessation of the attack, the centres respond extremely readily to irritation—a second attack being readily induced. But after from ten to fifteen minutes an increased resistance in the cells is to be observed.

B. Non-Motor Cortical Areas.

7. Irritation of non-motor parts of the cortex—posterior part—causes at first no convulsions, but after a latent period a general convulsion ensues. I need not point out how directly this bears upon the case we have had under consideration.

C. Influence of Destruction of Cortex.

8. Destruction of the motor areas on one side prevents irritation of the non-motor parts from inducing an attack. And irritation of the non-motor areas of the opposite side causes a general convulsion, confined to the side opposite the irritated area.

9. Any great destruction of the motor areas during an attack stops the convulsions in the muscles supplied.

10. A convulsion may be caused by irritation of the white substance from which the grey matter has been removed, but the convulsions stop without any clonic stage whenever the electrodes are removed. The convulsions induced do not tend to become general, and cannot be caused unless the current is stronger and only a small portion of the cortex has been removed. Irritation of the white substance lying below those parts of the cortex which are not motor causes no attack.

D. *Irritation of Medulla Oblongata.*

11. Irritation of the medulla oblongata causes a well-marked general muscular spasm, but no true epileptic attack.

E. *Influence of Bromide of Potassium.*¹

12. On administration of bromide of potassium in large doses, it is almost impossible to induce an attack by irritation of the cortex, while irritation of the subjacent white substance causes spasms as in the unpoisoned dog.

He concludes—

1st. The convulsive attacks which are induced in dogs by electrical irritation of the brain are the result of stimulation of the cortical centres, and present, according to the source of irritation, the greatest similarity to the so-called cortical, or to the idiopathic epilepsy of man.

2nd. That between the so-called cortical and idiopathic epilepsy there exists—in their pathogenetic aspects—no real difference; but from the facts that the first is really a symptom, the result of organic brain affection, and in its clinical course is not identical with the latter, it must be kept distinct from the latter.

3rd. The convulsive attacks of idiopathic epilepsy, as well as the attacks of petit mal, are the effects of a primary diseased condition of the cortex cerebri.

4th. The multiple character of the picture of the epileptic attack depends upon the different manner and degree in which the pathological cortical irritation, which is the basis of the epileptic attack, spreads.

5th. The theory which places the starting-point of the epileptic attack in the centres of the medulla oblongata and Pons Varolii—is in opposition to the clinical features of the attack, and is not based on sufficiently sure facts even in its relation to the elucidation of the epileptic attack.

Such experimental evidence, especially when supported by the older observations of Luciani² and of Bubnoff and Heiden-

¹ 'Neurol. Centralbl.,' 1884, No. 2.

² 'Sulla patogenesi dell' epilessia; Rivista sperimentale di Freniatria,' &c. 1878, iv.

hain,¹ is of the greatest possible value, and may be said to demonstrate beyond dispute the cortical nature of all epilepsies.

The many transitional forms of epilepsy uniting Jacksonian convulsions with the idiopathic disease are of importance, as still further bearing out the experimental and clinical evidence of the cortical nature of all forms of the malady.

On the one side we have cases such as Heslop's, where the disease is apparently due to direct pressure on, or at least to direct irritation of the cells themselves. In this class we must also place many cases of epilepsy from syphilitic tumours, and perhaps a few post-hemiplegic cases of epilepsy.

Very closely connected with this class we have another well-marked group of cases connected with tumours in the *white* substance of the hemispheres, but not directly involving the ganglionic parts of the brain. These tumours appear to produce changes in the ganglion cells indirectly through nerve fibres upon which they press. I need not here adduce evidence—now so well known to every one—that such changes are continually brought about in cells through the action of nerve fibres, which are, after all, but continuations of the nerve cells themselves.

Not far removed from such cases are the experimentally induced epilepsies caused by striking the heads of guinea-pigs in which small hæmorrhages among the nerve fibres appear to set up the epileptic state; and in close clinical relation to these experimentally induced fits are most of the cases of post-hemiplegic epilepsy, which apparently owes its origin, in the vast majority of cases, to the presence of small extravasations in the neighbourhood of areas which have had their blood-supply interfered with by embolism.

Passing now outside the encephalon, we have Brown-Séquard's epilepsy from section of the spinal cord and of nerves, irritative changes apparently spreading upwards along the nerve trunks to affect ganglion cells connected with them; and, closely corresponding to these again, we have the so common "fits" of childhood, which, though at first connected with difficult teething or obstinate enteritis, pass sometimes into true epilepsy.

¹ Pflüger's 'Arch.,' Bd. xxvi.

Most closely connected with true idiopathic epilepsy are those cases of patients who, having inherited a weak and mobile nervous system from their parents, yet remain healthy till some exciting cause sets up an epileptic condition. And it is interesting to observe that by far the most common cause in these cases is a sudden fright. The very close relationship of fear to certain muscular movements—the outward manifestations of fear—is well known, and renders it readily intelligible that a fright might so act on a weak set of ganglion cells as to establish in them an epileptic habit.

Lastly, we have true idiopathic epilepsy occurring in the children of epileptics—a set of cases exactly corresponding to the epileptic young begotten by Brown-Séquard's epileptic guinea-pigs.

ON A CASE OF AMNESIA.

BY E. A. DINGLEY, M.D. (LOND.).

House Physician to the Wolverhampton and Staffordshire Hospital.

I HAVE submitted the notes of this case of Amnesia to my friend and former teacher, Dr. Charlton Bastian. He agrees with me in regarding it as one of great interest, and as belonging to that category which has been described by him as due to "damage to commissures between the auditory and the visual word-centres." In his work on "The Brain as an Organ of Mind" (3rd ed., p. 642) he has recorded a case of amnesia, in which some of the principal defects seemed to be due to a lesion producing functional inactivity of the two sets of commissural fibres naturally existing between the auditory and the visual centres. Two or three pages further on he refers to an important case published by Dr. Broadbent,¹ in which there appeared to be damage to one of these sets of commissural fibres only (viz. the visuo-auditory). Many of the peculiarities of the present case are to be accounted for on the supposition that here, amongst other things, we have to do with some damage to the other set of commissural fibres between these centres (viz. the audito-visual). My case is therefore the complement of that published by Dr. Broadbent.

Thus, in Dr. Bastian's cases the patients could neither read aloud, nor write from dictation; in Dr. Broadbent's case the man wrote well from dictation, but could not read aloud; in the present case, the patient could read aloud fluently, though he could not write from dictation.

In regard to the fluency with which my patient read aloud, it will be well to remember also a remarkable case recorded by Dr. Hertz.² "This patient," says Dr. Hertz, "was able to articulate distinctly any words which either occurred to him

¹ 'Med. Chirurg. Trans.,' 1872 (Case viii.)

² 'Psycholog. Mag.,' vol. viii.

spontaneously, or when they were slowly and loudly repeated to him. He strenuously exerted himself to speak, but an unintelligible kind of murmur was all that could be heard. The effort he made was violent, and terminated in a deep sigh. On the other hand, *he could read aloud with facility.*"

The patient whose case is here recorded was admitted into the Wolverhampton and Staffordshire Hospital, under the care of Dr. Millington, and I have to thank him for allowing me to publish the following notes.

John C., *æt.* 56, a brewer's drayman, was admitted on March 27th, 1884, complaining of having had a "stroke." He was rather more intelligent than men in his position usually are, and could read and write well, and keep simple accounts. He had suffered from attacks of gout for some time, but had never had any other illness. On March 25th he complained of headache, but went to work, and seemed in fairly good health. On March 27th he went out to work as usual, but came home at 8 A.M. "looking strange," and got into bed with his clothes and hat on. After a time he got up, and walked about the house in a "delirious" condition. He could speak plainly, but his right arm was weak, and he dragged his right leg slightly.

On admission.—He was found to be a strong, healthy-looking man; his temperature was 99°, and his face flushed. He complained of frontal headache, and of a "giddy feeling." He was conscious and rational, but his mind seemed somewhat confused and dull; the functions of the cranial nerves were carefully tested, and were all found to be normal except the facial, which was markedly paralysed on the right side. He protruded his tongue without any deviation. His speech was thick and slow; he only answered questions, and did not volunteer any statements.

Deglutition was normal.

Both arms could be moved, but there was marked weakness on the right side.

He could walk, but the right foot was slightly dragged. Sensation was not affected.

As regards reflexes, his pupils reacted well to light, and also

to accommodation, and were equal; the epigastric and plantar were well marked, but the cremasteric was absent on both sides. Knee-jerk could easily be obtained in both legs, but not ankle-clonus. There was entire control over the sphincters. His arteries were rigid and tortuous, and his pulse had rather a high tension. The heart was slightly hypertrophied, but no murmurs could be heard. The arcus senilis was well marked. The digestive and respiratory systems were quite healthy. Urine, specific gravity 1012; no albumen.

March 28th.—Patient is less intelligent than he was yesterday, and even partly unconscious; he recognised his master, but said nothing to him, and had a very dazed appearance.

March 29th.—Patient is semi-conscious: he says "Thank you" to every question. He does what he is told, but seems very confused and dull. He talks incoherently in his sleep. His right arm is now as strong as the left, but the right facial paralysis remains well marked.

April 3rd.—During the last few days patient's intellect has cleared up considerably; he says a few simple words, and appears to comprehend pictures. He sometimes complains of headache, localised about the left temple and left side of the forehead.

April 5th.—Patient can recognise pictures, but he cannot name the objects represented; he uses circumlocutory phrases; as, when shown a picture of a camel, he said, "Egypt long way." *He can copy writing, and can write from dictation his own name and such words as "cat" and "dog," but not more difficult ones.* He attempts them, but only makes a scrawl, though he occasionally gets the first letter right.

April 10th.—Patient is still improving; he can name a few simple objects. His grip, as tested with a dynamometer, is 50 on the right side, and 45 on the left.

April 15th.—Patient is still making progress—he can now read aloud easy words, but stumbles over the longer ones.

April 26th.—Patient *can now read aloud very well*; he can copy writing and easy drawings, *but cannot write from dictation any better than on the 5th inst.* He is still unable to name the most common objects.

May 3rd.—Patient can now write a very fair letter asking

leave to walk in the garden, but cannot write the same words from dictation.

Remarks, with further details concerning Patient's condition two months after the attack.

This case is interesting, as being one of those which can be easily explained by Dr. Bastian's theory of Amnesia as set forth in his work on 'The Brain as an Organ of Mind' (3rd ed. pp. 600-690), to which and to his clinical lectures I am indebted for my information on this subject. Before considering the morbid condition present in this case, it will be advisable to describe the (theoretical) normal condition.

In our brains we may be legitimately supposed to have two centres, or cell and fibre mechanisms, which are specially concerned with the expression of our thoughts by speech and writing. The more important of these is called the "auditory word-centre," and in it are stored up, if we may so speak, the remembered sounds of words. So that when we wish to express our thoughts, the necessary words are revived in the auditory word-centre and are then uttered. That this is so, is evident from a study of the process of learning to speak; for during this period the child learns to associate, and does after many efforts associate, certain sounds which represent the name of an object with the object itself, and, later on, with the idea or remembered impression of that object. This theory is also endorsed by the fact that deaf children, who never hear word sounds, never reproduce them in speech.

The visual word-centre, in like manner, is concerned with the form or shape of the words and with their reproduction in writing. This I believe to be part of a larger "form" centre, which comprises and reproduces all visual impressions of form, size, colour, &c. That there is such a centre is, I think, indicated by the fact, that when we are trying to reproduce a sketch, we call up in "our mind's eye," as the saying is, a picture or remembered impression of the object, and copy this as we should copy the actual thing if it were present. Again, when we are in doubt about the spelling of a word, we often write down one or two variations and see which harmonises

most with our previously received impressions stored up in the visual word-centre.

Each of these word-centres may be stimulated to action in three different modes, viz. (1) by volitional impulses, as in spontaneous speech; (2) by associational impulses coming from the other centre, as in reading aloud and writing from dictation; and (3) by sensory impulses coming from the ear or eye, as the case may be, when repeating words or when copying them from a book. Each centre must, therefore, be supposed to be in relation with three ingoing sets of fibres.

In addition, each centre is furnished with outgoing fibres to the other centre; and with others going, in the case of the auditory word-centre, to the nerves supplying the speech muscles; and in the case of the visual word-centre, to those supplying the muscles used in writing: but before reaching these outgoing nerve fibres, the impulses are supposed to pass through a centre called the "kinaesthetic" centre, in which the sensations associated with different movements are received, and whose activity is supposed to help to regulate the execution of future movements of all kinds, including, of course, those concerned with speaking or writing.

Thus the auditory word-centre furnishes us with the words which we require in order that we may express our thoughts; and this, together with the kinaesthetic centre, furnishes, and in part regulates, the impulses to the various muscles concerned in speaking the words. In a corresponding manner, the visual word-centre would co-operate with its related part of the kinaesthetic centre, for the execution of the movements concerned in the writing of words.

As regards the relative importance of the auditory and the visual word-centres, the auditory evidently comes first, not only because speech is far more common than writing, but also because it seems likely that in health words are recalled in the visual word-centre only through the auditory word-centre; or if not entirely so, in very great part in this manner only. In the deaf and dumb, of course, the auditory word-centre is never developed, and words are revived only as visual impressions through the visual word-centre.

The position of these centres in the brain is considered by

Dr. Ferrier to be in the case of the visual in the angular gyrus, and in that of the auditory in the upper temporal convolution.

The centres may be damaged differently, so as to result in at least two kinds of defective action: (1) they may be paralysed, or (2) they may take on inco-ordinate action, *i.e.*, supply a wrong word instead of the right one.

I will now consider the modes in which we can (in accordance with a plan which I have learned from Dr. Bastian's lectures) test the efficiency of each centre, and apply them as I proceed to the case in question.

I. *Auditory Word-Centre.*

(1.) Is the patient's hearing good? This tests the condition of the sensory in-going fibres and of that part of the centre concerned with them. In this case the patient can hear perfectly well.

(2.) Can he understand speech? He comprehends everything that is said to him readily and well.

(3.) Can he speak? This tests the outgoing fibres and the portion of the centre connected therewith. But speech may be of three kinds.

(a.) Spontaneous: *i.e.*, can the patient express his thoughts as he wishes? Here, he can speak fluently and well, but cannot name objects (except a few familiar ones, and some which he has learnt since his attack). When asked the name of an object, he makes attempts to say the word, but cannot do so, and is quite conscious of his failure (paralytic amnesia). Sometimes, however, he says a word somewhat resembling the correct one in sound, as "spinnacles" and "stectacles" for "spectacles," or "rescern" and "concern" for "discern." At other times he will use a word, the meaning of which has a distant connection with the one required, as "mother" for "wife," and "brother" for "son"; or, again, he will use a periphrasis, as, when asked to name a camel, he said, "Egypt—a long way—go a long way—carry things—hot place." All the time he is quite clear as to the word he wants, and will recognise it immediately it is spoken, and will very accurately pick it out from a list of words resembling it in sound, or even from a list of synonyms.

Adjectives, verbs, etc., seem to present little or no difficulty, it being almost entirely noun-substantives that are unable to be recalled. Since the patient has been under observation he has learnt many names, and can recall others which formerly he could not do, and so has a fair vocabulary; but still he is constantly at a loss for a word in conversation.

(b.) Associational or mnemonic speech; or has the patient the power of repeating things from memory? This one has. He can count well, can say the alphabet, and can repeat the Lord's Prayer. In this case the bond of association or memory is so strong that it lasts uninjured after voluntary speech has been damaged, for the patient can say words under this influence that he cannot say voluntarily.

(c.) Imitative speech; or can the patient repeat words after you? This man at first could not repeat anything, but now he can repeat almost any simple word, with only an occasional difficulty. A very long word or a phrase he generally stumbles over more or less.

II. *Visual Word-Centre.*

(1.) Is the patient's sight good? If so, the in-going fibres are clearly intact; and in this case they are, as the patient can see perfectly well.

(2.) Does he comprehend printed or written words? He does very readily. He reads to himself very often, and obviously understands what he reads, since he can answer questions upon what he has been reading.

(3.) Does he recognise common objects? This man will recognise anything; can describe it, and knows all about it; but cannot name it.

(4.) Can the patient write? This tests the condition of the out-going fibres, and of the portion of the centre connected with them. Here, as in speaking, we have three varieties.

(a.) Can the patient write spontaneously? If so, the centre will respond to volitional impulses. In this case there is some defect, for although he can write simple sentences, he cannot write long or difficult ones, such as a description of an object.

(b.) Can the patient write things from memory? This is associational or mnemonic writing. Here he cannot very

well. He can write his name, but he has marked difficulty in writing the alphabet, and cannot write the Lord's Prayer, which he can repeat fluently; thus showing that here the defect is in the visual word-centre itself, and not that it is prevented from acting by a defect in the auditory word-centre.

(c.) Can the patient copy words—imitative writing? He can do so very accurately, copying either printing or writing, or transferring one to the other. He can also copy easy drawings exceedingly well, considering his abilities.

III. *Auditory and Visual Word-Centres Combined.*

We have two ways of testing whether these centres can act in unison, and therefore whether their commissural fibres are intact.

(1.) Can the patient read aloud? In doing this the visual word-centre receives a stimulus along its sensory in-going fibres, and passes it along the commissure to the auditory word-centre, where it calls up the corresponding sound, the stimulus to utter which passes along the out-going fibres to the kinæsthetic centre, and finally to the muscular nerves. This man *can read aloud with perfect ease, reading long and difficult words clearly and distinctly*. The fact that he can read aloud and yet cannot name objects shows that there must be a visual word-centre, as the auditory word-centre can be stimulated by the reception of impulses from it, but cannot be roused into activity by volitional impulses originated on seeing the object.

(2.) Can the patient write from dictation? This process is the reverse of the preceding, the sensory stimulus reaching the auditory word-centre, and going thence to the visual word-centre along commissural fibres belonging probably to another and a distinct set. In this case the patient has considerable difficulty in writing from dictation, only being able to write simple words, and not always these. Sometimes I have noticed that he was able to write a word spontaneously which he could not write from dictation, but this was not a constant or uniform condition.

We are now met with this problem: If he can read aloud, the commissural fibres must be intact; and if he cannot write

from dictation, they must be damaged. How can we reconcile these contrary propositions?

There are two explanations available :—

I. That there is a double set of commissural fibres—one the audito-visual, and the other the visuo-auditory; and, further, that one of these can be damaged and the other left intact.¹

II. That after some time the impulses from the visual word-centre, instead of going to the auditory and then to its related kinæsthetic centre, go direct to this latter kinæsthetic centre, passing by the auditory centre. But if the visuo-auditory fibres can be superseded like this, why should not the audito-visual be similarly superseded? In reply we can only make this suggestion, that the auditory word-centre having so much more to do than the visual, it would be an obvious advantage if it could be relieved from transmitting these commissural impulses by their going direct to the kinæsthetic centre. It seems possible that in some people this may be brought about by education and practice, though in the case of a man in the position of this patient, such an explanation would seem hardly likely to be the correct one.

To sum up, then, we find that in this case there is—

I. Some aphasia, though this is much less than it was.

II. Some paralytic amnesia, viz.,—

(a.) As regards the auditory word-centre :—

1. Diminished excitability to volitional impulses (spontaneous speech).
2. Normal excitability to associational impulses (reading aloud).
3. Diminished excitability to sensory impulses (imitative speech).

(b.) As regards the visual word-centre :—

1. Diminished excitability to volitional impulses (spontaneous writing).
2. Diminished excitability to associational impulses (writing from dictation).
3. Normal excitability to sensory impulses (imitative writing).

¹ On this subject see Dr. Bastian's 'Brain as an Organ of Mind,' 3rd ed. p. 640.

III. Some inco-ordinate amnesia extending to the same parts as are affected by the paralytic forms.

Therefore the lesion has damaged the auditory word-centre and the audito-visual commissural fibres, and to a less extent the visual word-centre.

As to the nature of the lesion, I should think that it was probably a thrombosis affecting a small area in the region of these centres as described above.

Note, October, 1885.—It is now more than eighteen months since the attack, and the patient is in very much the same condition. He has learnt some more words, and has a little more power in recovering names, but only after considerable delay and many failures. His general health is very good; his grip is, right 85, left 80; and there remains only a slight trace of the right facial paralysis.

ON A CASE OF BILATERAL DEGENERATION IN THE SPINAL CORD, FIFTY-TWO DAYS AFTER HÆMORRHAGE IN THE CEREBRAL HEMI-SPHERE.

BY W. B. HADDEN, M.D., AND C. S. SHERRINGTON, M.B.

EDWARD S., aged 63, an upholsterer, was admitted into St. Thomas's Hospital under the care of Dr. Stone, on March 5th, 1883.

The history was that he was found on the top of a tramcar, helpless, but not unconscious. He was at once taken to the hospital. Nothing more was ascertained about him, except that he had been under treatment at an ophthalmic hospital.

State on Admission.—Patient is conscious, and quite sensible. He cannot articulate, except to say "Yes" or "No" in answer to questions. He tries to speak, and can make sounds, but is unintelligible. He shuts his eyes when asked, and tries to put out his tongue. He can only protrude it very slightly, and it deviates to the right.

The mouth is drawn to the left. The right arm, which is rigid, is quite powerless, and sensation is impaired. There is also partial loss of motion and sensation of the right leg, but it is not rigid. The knee-jerk is brisk on the right side, and ankle-clonus is present. There is a slight elbow-jerk, but no wrist-jerk on the right side. The plantar reflexes are equal on both sides.

The lungs are emphysematous, and signs of bronchitis audible, mainly at the right base posteriorly.

The heart appears normal. The arteries are tortuous and thickened. There is no albumen in the urine. Ice was applied to the head, and four leeches to each temple. The bowels were freely acted on by croton oil.

March 9th—There is now no aphasia; but there is some difficulty in articulation, on account of the facial paralysis.

The tongue is protruded markedly to the right. The pupils are small, but equal. The bladder is distended, and he passes his water under him.

April 16th.—There is no return of power in the arm, and very little in the leg. The right hand is swollen, the skin over the backs of the fingers smooth and devoid of wrinkles, and the nails are more curved over the tips of the fingers than on the left side. There is some atrophy of the muscles of the thumb and of the extensors of the forearm. The elbow and knee-joints on the right side are stiff, and there is pain on making passive extension. The tendon-reflexes of both legs are brisk, but much more so on the right side. There is marked ankle-clonus on the right, but none on the left side.

The patient gradually got more and more drowsy, and albumen was found in the urine.

He died on April 26th, fifty-two days after the onset of the attack.

The temperature of the axilla on the paralysed side was sometimes half a degree or even a degree higher than on the left side, but occasionally it was equal on both sides, and rarely the temperature on the paralysed side was lowered. During the last nine days of life there was marked pyrexia.

Post-mortem Examination.—Body of an emaciated old man. Numerous Pacchionian bodies at the vertex of the brain on each side of the longitudinal fissure. The left hemisphere appears smaller and more flattened externally than the right.

The vessels at the base are gaping, and their walls are much thickened. There is no obstruction. There is moderate excess of the sub-arachnoid fluid.

Outside the forepart of the left lenticular nucleus some ochre-coloured clot is seen. Farther back, the clot is larger, the adjoining edge of the lenticular nucleus is lacerated, and the internal capsule between the caudate and lenticular nuclei is involved. The external capsule and claustrum cannot be distinguished. The hæmorrhage also involves the most anterior part of the lenticulo-optic segment of the internal capsule. Except for the laceration of the outer part of the lenticular nucleus, as mentioned above, the corpus striatum and optic thalamus are unaffected. The hæmorrhage measures

about an inch and a half, or rather more, in length, and involves nearly all the motor part of the internal capsule. The white matter forming the roof of the left lateral ventricle is softened and tinged yellow. The cortex everywhere is healthy. No signs of any other lesion in the brain.

There is no evident change in the crus and pons, but the left anterior bulbar pyramid has a slightly pinkish translucent appearance, in marked contrast with that on the opposite side. The degeneration is also visible throughout the entire length of the right lateral column in the cord. It is most marked in the cervical region, less evident in the dorsal; but in the lumbar portion it again becomes very distinct, and reaches up to the periphery of the cord.

There is no difference between the median nerves on the two sides. The lungs are emphysematous, the right side of the heart dilated and hypertrophied, the liver fatty, and the kidneys small and very granular.

In sections prepared from the hardened cord and pons, the following condition is observable.

Pons and Medulla Oblongata.—The pyramid of the left side gives a decidedly larger area on cross-section than does that of the right side. There is no obvious difference as regards depth of stain between the right and left sides, either in preparations coloured by carmine, or after Weigert's hæmatoxylin method. Under the microscope the left pyramid proves to be very much degenerated; the right pyramid appears to be undegenerated, save in a small portion of it, lying ventrally and medially, adjoining the grey matter of the "nucleus of the arciform fibres" (Schwalbe). The area given by the degenerating tracts at the level of the lower ends of the inferior olivary bodies is indicated in Fig. 1. The depth of the stippling is intended to be proportional to the degree of degeneration. It is notable that the degenerated area approaches the olive very closely, and that upon the left side three small islets of degenerated fibres lie dorsal to the main pyramidal mass, within the *formatio reticularis*.

Spinal Cord.—The right lateral column is slightly smaller than the left, its smallness being due to diminution of its posterior two-thirds, the bold outline of that part on the left,

is on the right replaced by a flatter line. Fig. 2 represents the state of things opposite the fifth cervical nerve-root. Here, in sections stained with logwood, or with carmine or picrocarmine the area of the right crossed pyramidal tract is coloured a little more deeply than normal. This is more obvious in carmine than in logwood preparations, and is most obvious in that part of the tract which borders the direct cerebellar band. No change, however, is obvious in either direct pyramidal tract, or in the left lateral column. In sections stained after Weigert's hæmatoxylin method¹ a degenerative change is at once obvious to the naked eye in both lateral columns and on both sides of the anterior median fissure. On the right side the area of the crossed pyramidal tract is tinged with brown, contrasting with the inky tint of the rest of the antero-lateral column. The region of the direct pyramidal tract also is brownish, and not merely to left of the anterior fissure but to right of it. Further, on the left side the area of the pyramidal tract in the lateral column is brown—though not so strikingly so as on the right. Lastly, the periphery of the antero-lateral column from lip of anterior median fissure round to some millimeters behind the anterior nerve-roots offers a brownish zone, a half or more of a millimeter deep in the section—the method even in sections from well-hardened cord causes much shrinking.

The degree of change in the right half of the cervical cord is greater in the lateral column than in the anterior; in the left half it is greater in the anterior column than in the lateral.

Both in carmine-stained sections, and in those stained by Weigert's method, close examination shows the finer anatomy of the degeneration to be as follows:

In the right crossed pyramidal tract there is some excess of connective tissue, especially of large deeply-staining cells which lie between the cross-sections of nerve-fibres. Individual nerve-fibres are not so distinct and easily countable as normal, although many are perfectly distinct. Many of the stromal spaces are vacant; presumably because the cross-sections of the fibres have dropped out in the process of preparing the specimen for the microscope. In some of the stromal spaces

¹ 'Fortschr. der Med.,' April 15, 1885.

lie large coarsely granular, rather highly refracting, and ill-stained, rounded bodies (cf. Fig. 7 *d*). They are not deeply coloured by carmine or Weigert's hæmatoxylin method. A nucleus is not certainly evident in them, but with carmine one part of them often colours more deeply than the rest. Whether they are similar to the "granulation corpuscles" discussed and figured by Bastian,¹ or whether they are portions of much altered nerve-fibres, is not clear to us. Some stromal spaces are filled by ill-defined palely-staining homogeneous masses; some contain simply an almost uncoloured granular *débris*; occasionally are seen cross-sections of fibres to all appearance healthy, but that the axis-cylinder stains more deeply than normally and appears enlarged. Without more exhaustive examination, the impression left upon us is that the fibres degenerating, although not of the largest of the antero-lateral column, are of the larger rather than of the smaller—but are not all of equal calibre.

Similar changes are present in the left lateral column, but there the increase of connective-tissue elements is very slight. In the anterior columns, as in the left lateral, the changes are almost confined to the nerve-fibres, the stroma not being obviously thickened. In the dorsal cord, Fig. 3, at the level of the second nerve-root, the bilateral character of the degeneration is evident. The change is still most marked in the right lateral and left anterior columns; the shape of the area involved is not much different from that of the cervical region.

Fig. 4 shows the change at the level of the 10th dorsal nerve, here again evidently bilateral; but of somewhat altered shape. The area of the direct cerebellar tract has been encroached upon, and as the cerebellar fibres are here scattered, and are set in much tissue continuous with the deeper pial layer, a little care is wanted not to suppose that the degeneration-field here extends to the periphery of the lateral columns from the posterior root forwards. At this level no degenerating fibres are observable at the right border of the anterior fissure.

In Fig. 5, opposite the 2nd lumbar nerve, it is seen that the

¹ Trans. of Medico-Chir. Soc., vol. I. p. 528. Pl. XI. Fig. 20.

degeneration area lies further outward from the central grey matter. Degenerating nerve-fibres still are present in the left border of the anterior fissure.

In Fig. 6, at the 1st sacral root, the degeneration is still bilateral in character, but absolutely confined to the periphery of the lateral columns, as a triangular area, more obvious on the right side.

The median nerve of the paralysed right limb was healthy.

The above case is interesting, from the opportunity offered for microscopic examination of the secondary degeneration in the cord, so early as seven weeks after the occurrence of the cerebral lesion. That the degeneration in the cord was due to the hæmorrhage which occurred fifty-two days before death, there is no good reason to doubt. It is noteworthy that the degeneration in this early stage shows very little sclerosis; even in the crossed pyramidal tract in the right lateral columns where it is most, it is quite slight. It has been shown that in the dog the amount of sclerosis in the degeneration patch is proportional to the age of the degeneration, *i.e.* the farther the degeneration dates back, the more the fibrous tissue in the track of degeneration, until a period is reached, within the year in the dog, at which the only evidence of a degeneration-process that has occurred is the scar it has left behind. By this criterion a glance at a carmine-stained section of the cord from the present case would show that death had followed at a comparatively short period the initial lesion in the brain.

The process of degeneration, in the present case, seems as far advanced in the lumbar region as in the cervical. This with the early date is interesting, and agrees with observations to the same effect in the dog.¹ In fact the name "descending" degeneration given to the secondary degeneration of nerve-fibres distal to the seat of initial lesion seems scarcely justified. It implies an idea of change observably extending downward along the fibres, whereas there is great dearth of observation of such a progression. *A priori* we conceive there is as much reason to suppose the change an ascending one, or one occurring simultaneously throughout the length of a fibre, as to imagine it descending. Of the fibres in the spinal cord

¹ Sherrington, 'Journ. of Physiol.' vol. vi. p. 177.

trophically dependent on the brain, the earliest portions to suffer after brain injury might well be supposed those at greatest distance from their trophic centre. Were speculation useful here, many arguments suggest themselves for and against each of the three views. But we would remark that serious objection against the name "descending" degeneration can be urged, until more observation of its descent are forthcoming.¹

The Figures 1 and 2, to which references will be found on p. 511, show a point on which we would lay stress; the evidently large diminution which the degenerating tract undergoes in amount as it passes from the medulla oblongata into the spinal cord, in the region of pyramidal decussation. Into what structures do the manifold degenerated fibres stopping short at this level embouch?

A point of great interest in the present case is that a degeneration doubly bilateral in the spinal cord has resulted from injury confined to one hemisphere of the brain. Pitres has described such cases.² In the dog analogous cases have been observed by Moeli,³ by Löwenthal,⁴ and by one of us.⁵ Schäfer⁶ also, as long ago as 1883, noticed that in the spinal cord of a monkey operated on by Drs. Ferrier and Yeo, the cervical enlargement contained a degeneration in the lateral column on the same side as the cerebral injury.

In the present case, the degeneration in the lateral column on the same side as the hemisphere injured, when compared with that of the crossed pyramidal tract opposite, presents the same characters as in the dog; it is more diffused, less defined, with edges softer. Although the difference between the degenerations on each side of the anterior fissure is not great, still differences the same in kind are present there also, the right-hand degeneration being more diffuse. In the dog, one of us was led to believe that the degeneration on the same side as the cerebral injury either appeared rather later, or, if it began simultaneously with that of the crossed tract, was at first quite slight, and only later, when that of the crossed tract had

¹ The only support we know of for the term is by Schiefferdecker. Virchow's 'Archiv,' 67.

² 'Archives de Physiologie norm. et path.,' vol. v. 3rd series.

³ 'Archiv. f. Psych.,' vol. x.

⁴ 'Zeitschrift Zoologie,' Geneva, 1885.

⁵ 'Jour. of Physiol.,' vol. vi.

⁶ 'Jour. of Physiol.,' vol. iv.

already been a little time in full play, reached its own stage of full activity. If one may from amount of sclerosis present judge the age of a degenerative lesion, one would say that in the present instance the lesion in the right lateral column had been in existence longer than that in the left. In the dog too, especially when the degenerative lesion is small, it seems that the degeneration on the same side as the cerebral injury does not progressively diminish in quantity from above downward, as does the lesion of the crossed pyramidal tract. In the case before us, the left-hand degeneration does not at any lower point in the spinal cord seem larger than at some point more proximal. The only hint of such a character is in the comparison of sections from the levels of 3rd and 5th cervical nerves respectively; the degeneration in the left lateral column may be slightly more extensive at the 5th than the 3rd.

Pitres does not find any degeneration in the pyramid of the side opposite the cerebral lesion. It must be remembered that Pitres is always dealing with the scar left by the degeneration rather than with the degeneration itself. His earliest case is one of more than six months' standing. It seems doubtful whether the degeneration of a small number of fibres scattered somewhat sparsely through a tract of uninjured fibres would leave any scar—at least, any scar detectable by present methods of observation. In the present case, as above stated, we believe the degeneration to be bilateral in the medulla oblongata, although extremely asymmetrical there—much more asymmetrical than at any point in the spinal cord below the decussation of the pyramids.

There are some points of clinical interest in this case to which we would briefly call attention. The rigidity of the paralysed limbs occurred at the onset of the attack, and persisted until death. It is usually asserted that early rigidity in hemiplegia is due to irritation of the pyramidal-tract fibres, and this assumption receives some support from the present case. Whatever may be the explanation, there is no doubt that the so-called early rigidity in some instances remains permanent, no interval elapsing between it and the slow contracture which results from secondary spinal degeneration. We have for some time had a case under observation, in which

rigidity of the paralysed arm and leg ensued immediately on the attack. A year later the rigidity was still present in a very marked degree.

The early occurrence of ankle-clonus in our case deserves some notice. This we have observed in other cases in which no obvious rigidity was present. Attention has also been directed to this subject by Pitres.¹

Although, in the case we have narrated, the secondary degeneration was bilateral, it is interesting to note that rigidity and ankle-clonus were not present in the non-paralysed side. The only indication of any organic change was an exaggeration of the knee-jerk on that side.

On this point, Pitres, in the paper to which we have already alluded,² says: "Bilateral exaggeration of the knee-jerk and bilateral contracture of the lower limbs may exist in cases in which the secondary sclerosis is perfectly unilateral; and, on the other hand, they may not be present when the secondary sclerosis occupies both lateral columns."

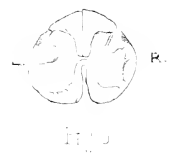
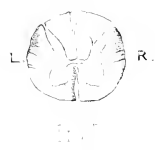
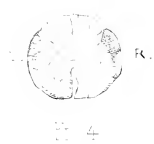
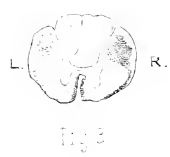
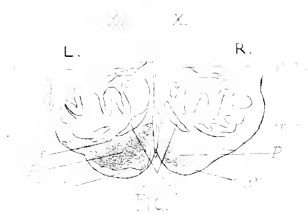
A similar opinion had already been expressed by one of us on theoretical grounds.³

It was then suggested that the occurrence of ankle-clonus and rigidity on the non-paralysed side in hemiplegia might sometimes depend on irritation conveyed from the motor cells on the hemiplegic side to those on the opposite side of the spinal cord. The intimate connection between the nerve-nuclei which preside over the lower limbs, and the comparative independence of the motor nerve-cells controlling the movements of the upper limbs, explain many facts of the highest clinical interest.

¹ 'BRAIN,' 1884, p. 310.

² 'Archives de la Physiologie norm. et path.' 15 Février 1884.

³ Hadden, in 'St. Thomas's Hospital Reports,' 1882.



1897

1897

EXPLANATION OF PLATE.

Abbreviations:—

in. ol.	..	inferior olivary body.
P.	Pyramidal tract.
sub. ol. t.	..	subolivary tract (olivenzwische schicht).
a. ol.	..	accessory olivary bodies.
n. ar.	..	nucleus arciformis.
R.	right-hand side.
L.	left-hand side.

Fig. 1. Cross-section through medulla oblongata, opposite the lower ends of the inferior olivary bodies.

„ 2. Ditto, through the spinal cord opposite the 5th cervical nerve root.

„ 3. Ditto, ditto, 2nd dorsal nerve.

„ 4. Ditto, ditto, 10th dorsal nerve.

„ 5. Ditto, ditto, 2nd lumbar nerve.

„ 6. Ditto, ditto, 1st sacral nerve.

„ 7. Portion of the region of the right crossed pyramidal tract at the level of the 10th dorsal nerve, at the spot marked * in Figure 4. Zeiss—Objective F. Ocular 2, drawn with camera. Preparation coloured by Beale's carmine-fluid; mounted in Canada balsam.

a, normal nerve-fibres seen in cross-section.

b, nuclei of neuroglia cells.

c, capillary blood-vessel.

d, stromal space in which the nerve-fibre has degenerated—occupied partly by a coarsely granular, palely staining, non-nucleate mass.

e, stromal space empty, perhaps because its nerve-fibre was degenerated.

All the figures except the last are magnified two diameters. In the lithographic reproduction, especially of Figs. 2, 3, 4, 5, the stippling, indicating the areas of degeneration, is much darker than in the original drawings.

THE INFLUENCE OF THE INTENSITY OF THE STIMULUS ON THE LENGTH OF THE REACTION TIME.

BY JAMES MCKEEN CATTELL,

Assistant in the Psychological Laboratory, University of Leipsic.

DURING the past two years, Dr. G. O. Berger and I have been carrying on a series of experiments in the Psychological Laboratory of the University of Leipsic, looking to determine the relation between the intensity of the stimulus and the length of the reaction time. Wundt, Exner, and others had already made experiments on this subject, but it seemed to need a more thorough investigation.¹ We undertook to determine the influence of various intensities of the electric shock, and of light on the length of the simple reaction time, and on the reaction time complicated by the addition of simple cerebral operations.

The term "reaction time" is now generally understood. If one lifts one's hand as soon as possible after the sudden appearance of a light, the interval between the application of the stimulus and the beginning of the muscular contraction is a reaction time. In order to investigate the influence of various intensities of light on the length of this time, we used a light produced in a Geissler's tube by an induction current from six Daniell cells. This light we took as normal, and kept constant. We then arranged five weaker intensities by putting smoked glass before the light. The amount of light transmitted through the smoked glass we determined photometrically. If we set the intensity of the normal light $VI = 1000$, then the intensities of the lights would be

I.	II.	III.	IV.	V.	VI.
1	7	23	123	315	1000

We further obtained two still brighter lights (vii and viii) by

¹ Wundt, 'Physiologische Psychologie,' 2. xvi.; Exner, 'Hermann's Physiologie,' 2. 2. iv.; Kries u. Auerbach, 'Archiv f. Anat. u. Physiol.,' 1877; Vintschgau u. Hönigschmied, 'Pflüger's Archiv,' xii.; Wittisch, 'Zeitschr. f. Rat. Med.,' xxxi.

means of lenses, but could not determine with our photometer the relation of these to the normal intensity. The observer sat in the dark, and looked through a telescopic tube at the point where the light was to appear. The following table gives the average of 150 reactions made by each of us with the several intensities. No reactions at all were omitted in taking the average. The second line, marked M, gives the average of the variation of each from the average of all the reactions; that is, if A is the average of n reactions a, b, c, d , then

$$M = \frac{(A-a) + (A-b) + (A-c) + \dots}{n}$$

all the differences being taken as positive. M shows us how much the reactions differ from one another, and when we know the number of reactions, we can find the probable error of the average. In the table .001s. is taken as the unit of time.¹

TABLE I.

Intensity.	B.								Average.
	I.	II.	III.	IV.	V.	VI.	VII.	VIII.	
Time.	308	235	208	200	192	195	177	168	210
M.	26	18	16	15	15	17	18	16	18

C.									
Time.	251	175	160	148	147	143	135	128	161
M.	30	17	16	14	15	13	16	19	18

It will be seen from the table that when the light is taken very weak, just strong enough to be seen, the times are the longest and (with one accidental exception, B between v and vi) the greater the intensity of the light, the shorter the time of the reaction. I cannot, however, formulate a general law from the table.

In substantially the same manner the relation between the

¹ It should be mentioned that in our first experiments the absolute time is not certain. The times were measured with aid of an electro-magnet, and we assumed that when a current was sent through the coil, the armature was attracted instantaneously. This is not the case, and I have made an allowance for the error, but the times may be as much as .01s. wrong. At all events the relative times are correct, as the error was constant.

strength of an electric shock and the length of the reaction time was determined. The shock was received on the left forearm, and the reaction made with the right hand. We used four intensities; the strongest, IV, somewhat painful, the weakest, I, just enough to be felt. The two intermediate intensities made up, as far as we could judge, four equal steps. The averages of 150 reactions by each observer and on each intensity are given in the table.

TABLE II.

Intensity.	I.	II.	III.	IV.	Average.
B.					
Time.	182	163	158	160	166
M.	17	14	12	11	13
C.					
Time.	164	155	132	131	145
M	19	18	14	14	16

It will be noticed that with the electric shock, as with light, the time of the reaction becomes shorter as the stimulus becomes stronger. The differences are not, however, so great, and for the intensities III and IV, the times are about the same; with IV the reaction was probably retarded, because the shock was painful.

In connection with the experiments on the intensity of the light, we made others to determine whether or not the quality of the stimulus, that is, the colour of the light, has any influence on the length of the reaction time. The averages of 180 reactions made by each of the observers, and on each of the six colours used, are given in the table.

TABLE III.

	White.	Red.	Yellow.	Green.	Blue.	Violet.	Average.
B.	196	203	192	196	199	201	198
C.	155	162	160	156	161	153	158

The table does not show any decided difference in the times for the several colours. Violet and green, which I have found (see last number of 'BRAIN') must work longer on the retina than the other colours, in order that a sensation may be called forth, do not seem to cause a longer reaction time; this is because the reaction is made on the light, without waiting until the colour has been distinguished.

The time is longer when it is necessary to distinguish the colours before the reaction is made. We can determine this time, if instead of always reacting as quickly as possible we use two lights of different colours, say blue and red, and let the subject react only on one of them. The subject does not know which light is to come, but is to lift his hand as quickly as possible if it is red, but not at all if it is blue. We thus add to the simple reaction the time it takes to see whether the light is blue or red, and complicate somewhat the process of volition in the simple reaction time. We can further let the subject lift his right hand if the light is red, his left hand if it is blue; we then have, besides the time necessary for the simple reaction and for distinguishing the colour, the time it takes to make a choice between two motions. The results of experiments made with three intensities of light (V, III, and I) are given in the table.

TABLE IV.

	B.			C.		
	V.	III.	I.	V.	III.	I.
Reaction time	189	218	273	189	209	303
Reaction with Perception time	238	293	373	274	328	417
Reaction with Perception and Will time	287	320	393	356	388	495
Perception time	49	75	100	85	119	114
Will time	49	27	20	82	60	78

It seems from the table that the time it takes to see or perceive a colour becomes shorter as the intensity of the light becomes stronger, but that the will time is not a function of the intensity of the stimulus.

Clinical Cases.

A CASE OF AMYOTROPHIC LATERAL SCLEROSIS WITH CLONUS OF THE LOWER JAW.

BY C. E. BEEVOR, M.D., M.R.C.P.,

*With a NOTE on the JAW-JERK, or MASSETERIC TENDON REACTION,
in HEALTH AND DISEASE.*

BY A. DE WATTEVILLE.

As Dr. Gowers has called my attention to a case published in America, in which an increased tendon-reflex of the muscles of the lower jaw was obtained, I have thought that this case, which I saw four years ago when resident at the National Hospital for the Paralysed and Epileptic, might be of interest.

The case was under the care of Dr. Ramskill, whom I have to thank for allowing me to publish it.

The patient, a woman aged 46, with no previous or hereditary history of importance, noticed one morning, nine months before admission, that the left side of her mouth felt "dead"; six weeks later the left arm, one morning, felt numb, and she could not hold objects with it; the forearm gradually got worse, and has been paralysed for five months; symptoms of bulbar paralysis gradually came on; in four months from onset her speech became affected, and in eight months swallowing was difficult. Seven months from onset, the right hand began to be weak.

On admission, she had all the signs of bulbar paralysis: difficulty in showing the teeth; inability to swallow, with regurgitation of fluid through the nares; inaction of the soft palate; speech unintelligible, only being able to make a moaning sound; her tongue she could not protrude past the teeth, although it was not much wasted; the masseters and temporal muscles were very weak.

In the left upper limb the small muscles of the hand, and the forearm were much wasted, and the wrist and fingers quite powerless; there was less wasting of the upper arm and shoulder, and very slight movement was possible in the left elbow and shoulder-joints.

In the right arm the ball of the thumb and interossei were

somewhat wasted, and the movements of the whole arm were weak.

In the left arm there was considerable rigidity of the joints, and this was present to a less degree in the right arm.

With regard to the lower limbs, they were not wasted; she could walk well, and the only thing remarked was some rigidity about the joints.

The *knee-jerks* were excessive on both sides, perhaps the left more than the right; but there was *no ankle-clonus*.

On filipping the lower end of the radius of either side, a very active contraction of the flexors of the elbow was produced.

The symptom which I think the most interesting in this case is the *clonus of the lower jaw*; this could very readily be produced by placing the finger on the teeth of the lower jaw and then depressing it, when immediately the muscles which closed the jaws passed into a state of clonic contraction, and the lower jaw vibrated as long as the pressure was kept up by the finger on the teeth; in fact, it behaved in the same manner as the heel does when ankle-clonus is produced. Clonus could also readily be produced by striking the masseters; and also if the zygomaticus major was percussed, retraction of the corner of the mouth ensued.

I do not know whether a similar case has been observed, but I do not remember to have heard of any one having been reported.

It is interesting to notice that the sclerosis of the cord was confined chiefly—if not almost entirely—to the medulla and upper part of the spinal cord, so that clonus of the lower jaw and radius tap-contractions were very readily obtained, while, on the other hand, ankle-clonus, which was tried for on several occasions, was absent, and though contraction of the extensors of the knees could be obtained by pressing down the patella from above, this method failed to produce clonus of these muscles.

According to the patient's account, the loss of power in the left arm occurred suddenly; but whether this was really the case, it is difficult to decide.

Since seeing this case, it has frequently occurred to me whether the chattering of the teeth produced by cold or by a rigor could be merely a clonus of the muscles of the jaw, and whether in these conditions the "tendon-reflexes" of all the muscles of the body are increased; as this is only thrown out as a speculation, I shall certainly test the tendon-reflexes in any case of rigor I may happen to see. From my personal experience, I think that the knee-jerk is increased after a prolonged stay in a cold bath; but I have not as yet been able

to produce ankle-clonus, though I have heard on good authority of its occurrence in a healthy individual under such conditions.

I may add that the patient soon returned home, and I lost sight of her, and I am unable to give details of her further progress.

NOTE.

As it does not appear to be generally known that a "jaw-jerk" can be readily elicited by an appropriate stimulus in most healthy persons, I take the opportunity offered by the publication of Dr. Beevor's observation to call the attention of neurologists to this point. The phenomenon is clearly of the same nature as that of the "knee-jerk," and is due to the sudden stretching of the masseter and other muscles of mastication. Hence the name I have ventured to give to it, in preference to the longer and less accurate term mandibular (or masseteric) tendon-reaction (or reflex).

The method of producing the contraction, which I find most effectual and convenient, is to introduce into the experimentee's opened mouth a tongue depressor or a paper knife, which is made to press with its flat surface upon the teeth of the lower jaw. It is better to place it somewhat laterally upon the canine and premolars than in the middle line upon the incisors. The blade is held down with a firm pressure, care being taken that the jaw is not fixed by any undue muscular contraction; the experimentee should just resist the strain downwards, and nothing more. A sharp tap with a convenient object, such as a ruler or a thin-bound volume, but best of all with a percussion hammer, is then struck upon the paper-knife, close to the teeth. A contraction of the stretched muscles then occurs of more or less amplitude, but which is usually quite appreciable to a finger introduced between the molar teeth on the opposite side.

In many cases, especially in such where one finds the usual tendon-reactions exaggerated, one readily obtains a very lively jaw-jerk. This fact, I feel bound to state, was quite familiar to me before it came to my knowledge that the phenomenon had been observed in a case in America. I have not been able to ascertain the author of, any details about, nor the reference to, this observation.* But it is to Dr. Beevor's mention of the occurrence of jaw-clonus in the case he now publishes that I owe to have had my attention drawn to the mandibular tendon-reactions.

When exaggerated, the jaw-jerk may be elicited by using the fingers to depress and medietely percuss the lower jaw. The left forefinger is placed either upon the teeth, according to the plan already described, or outside upon the projecting mental ridge, and the right index and middle finger used instead of the hammer.

* Both Dr. Gower's and Dr. Beevor's information concerning this case is limited to the bare fact of its existence.

Or, again, the hammer may be made to strike the jaw directly, on either side of the chin. I have thus, in one way or another, obtained evidence of what appears to be abnormal excitability in the nervo-muscular apparatus of mastication in various conditions, such as organic or functional nervous disorders, in convalescence from acute diseases, &c. Abnormal diminution, or absence, of jaw-jerk, on the other hand, is a subject which I prefer to pass over for the present, and reserve for a future occasion, when my experience of the variations compatible with health is more precise and extensive. It is possible that in time the condition of the jaw-jerk may acquire a diagnostic value in cases of bulbar or other disease. With reference to jaw-clonus (which it is the credit of Dr. Beevor to have been the first to describe in the present article), I may mention that I have, since he told me of it, demonstrated its occurrence, both in purely functional nerve disorder (as in a case of hysterical, tonic, and clonic spasms of the limbs, spontaneous and provoked), and in organic disease (as in a case of right hemiplegia with left hemiparesis, articulatory disturbance and ocular troubles). The methods for eliciting the clonus are the same as those just described for bringing about the simple jerk.

In conclusion, I will state that I have endeavoured, with the kind assistance of my friend, Dr. Waller, to obtain myographic tracings of the jaw-jerk, in order to determine the latent time of the phenomenon. We also attempted to record, on the revolving cylinder, the rhythmical movements which constitute the jaw-clonus. Our results give very nearly .02 second for the latency of the contraction of the muscles of mastication, when excited by percussion of the jaw according to the method just described. We found this period to be the same, both in the healthy and the diseased subject. Should these measurements be confirmed by further investigations, they will furnish another argument in favour of the view, that the so-called "tendon-reflexes" are phenomena due, not to a true reflex excitation, but to the direct stimulation of the muscles by the sudden extensile impulse of the blow. Dr. Waller, in some recent experiments, has determined the latency of the closure of the eyelid to the stimulus of a strong light. This process, the shortest true reflex known, occupies more than twice as long as the jaw-jerk, viz. about .05 of a second.

A. DE WATTEVILLE.

PRIMARY SPASTIC PARALYSIS AND PSEUDO-HYPERTROPHIC PARALYSIS IN DIFFERENT MEMBERS OF THE SAME FAMILY, WITH PROBABLE HEREDITY IN BOTH.

BY R. W. PHILIP, M.A., M.B.

Assistant to the Professor of Practice of Physic, University of Edinburgh.

THE following cases form a family group of considerable interest and importance. For the opportunity of studying them I am indebted to the kindness of Professor Grainger Stewart, under whose charge they have lately been, in the Royal Infirmary, Edinburgh.

The group consists of a father, G. P. sen., and two sons, A. P. and G. P. jun. As the family history is common property, I shall, before describing the individual cases, narrate so much of this as seems important. The father of G. P. sen. died about the age of sixty, of apoplexy, and the mother, aged fifty, from gradually advancing debility. Of this marriage, there were nine children, three daughters and five sons, beside the patient. Of all these the history is good. Uncles and aunts on both sides were numerous, and are reported to have been strong and healthy. G. P. sen. married thirty-two years ago, and his wife is alive and strong, aged fifty-three. The wife's family history is less perfect. A female cousin, aged fifty, has been so completely paralysed, as to be unable to feed herself for eighteen years, while an uncle, aged sixty-six, has been similarly paralysed for a number of years. By this marriage also there were nine children, whose individual history is of interest:—(1) girl, drowned accidentally, aged seven; (2) girl, alive and healthy, aged twenty-eight; (3) boy, died, aged fifteen, apparently of pseudo-hypertrophic paralysis. The father says that "for several years he hadn't the use of himself;" (4) boy, died aged eleven, with symptoms of dropsy; (5) girl, died, aged four, of scarlatina; (6) boy, alive and healthy, aged eighteen; (7) boy, died, aged fifteen, of pseudo-hypertrophic paralysis. His case was de-

scribed as such by Dr. Macphail, Whifflet,¹ (8) boy, A. P. (*vide infra*); (9) boy, G. P. jun. (*infra*).

Of the three at present in hospital,—

G. P. senior, aged sixty, was admitted on account of inability to walk properly. He has worked as a miner since he was little more than twenty, for the first twenty-five years among ironstone, latterly among coal. His occupation, besides taxing his strength much, involved continued working on his side, in cramped and constrained postures, often partially below water for hours. The space in which he worked not unfrequently measured from 2 feet to 2 feet 10 inches in height. Twelve or thirteen years ago, he met with a severe accident. The guiding rope for the hutches or low waggons, used for carrying coal from the cutting to the bottom of the shaft, broke, and his clothes were entangled by the rapidly passing hutches. He was dragged for about 100 yards, being much squeezed, and sustaining severe injury to the right leg and hip. In consequence of this he was laid aside for twenty-six weeks. On recovering, he kept to coal-mining, as being lighter, though the position necessary for work was more cramped. At this time he began to experience a sensation of stiffness in the right lower limb, which made the bent posture difficult. He also noticed that when he put his foot to the ground, the whole limb began to shake. He occasionally suffered from darting pains, described as “burning,” which passed up from the sole of the right foot to the loins. These gradually grew more constant and the general condition worse, till, two years ago, he had to give up work altogether, from inability to walk. In addition to the increased stiffness and pain, his right foot tended to drag and trip easily. He had great difficulty in extending the leg at the knee and hip, and this difficulty made him walk on his toes. The tendency to trembling of the limb grew more marked. A year ago, the left leg showed signs of becoming similarly affected, and during the last two months he has felt a cold, aching pain in the upper arm, during movement. Three days ago he began to experience pains in the right shoulder, and the “startings,” previously experienced in the leg, threaten to appear here also. The patient’s home surroundings have been comfortable. He has always had good food. He frequently got tipsy on pay-nights. At other times he was sober. There is no history or suspicion of syphilis.

Present condition.—Patient is a man of 5 feet 9 inches, of fair development and muscularity. Scattered over his face and extremities are numerous pigmentation spots and cicatrices, the remains of injuries received from time to time in the course

¹ ‘Glasgow Medical Journal,’ July, 1882.

of his work. The lower part of the thorax projects abnormally, and is separated from the abdomen by a well-marked horizontal depression. A certain degree of lordosis is present.

In standing, he stoops a good deal, and supports himself entirely on the left leg, the right being flexed at the knee and hip, and forcibly adducted, so as to cross its fellow. The left leg is slightly bent at the knee and adducted. Talipes equinovarus is not marked. In walking, he makes use of a stick in his left hand, and in his right, a triangle stool of appearance like Δ , the vertical side being held next himself, so as to afford a broad basis of support on the side towards which he tends to fall. As he lies in bed, the right thigh is flexed and adducted, and the right leg is flexed at the knee, the left to a less extent. The muscles of the right thigh, and the lower part of the abdominal muscles of the same side, show a high degree of contracture, while the calf muscles are flabby. On the left side, the muscles of the thigh, especially the hamstring and the adductors, feel rigid. Regulated movements of the lower limbs are practically abolished. The upper limbs show no lack of power of co-ordinated movement. Ankle-clonus can sometimes be elicited, but the rigid condition of the parts considerably interferes with its demonstration, as also with the knee-phenomenon. Over other tendons, however, a slight tap produces exaggerated movement. This is specially well marked at the elbow. There is no important alteration in the organic reflexes, though the initiation of micturition is delayed. The superficial reflexes are normal. The affected muscles exhibit a diminished reaction to faradic stimulations. The reaction to galvanisation is generally lessened, but the right hamstrings exhibit increased irritability, being also abnormally sensitive to mechanical stimulation. There are no qualitative changes. An aching pain and a sensation of stiffness is experienced in the right knee, thigh, hip and groin, and occasionally in the shoulder and neck towards the same side. Patient also describes a feeling of coldness and numbness which extends from the right foot to the corresponding thigh. Sensibility is unimpaired. The special senses are intact, with the exception of sight, which has been much less acute since the accident twelve years ago. There is no ophthalmoscopic change. Of trophic disturbances, besides the wasting of certain muscles there is to be noted the presence of a number of bald patches—true alopecia areata—scattered over the head, and of erythematous and scaly blotches about the feet. His memory is not good, and he is apt to make contradictory statements, so that the sifting of the history has been matter of difficulty. In other respects, the cerebral functions seem normal. Percussion over the spine reveals the presence

of a fairly well-defined sensitive area extending from the 7th to the 11th dorsal vertebra, and less distinctly as far as the sacrum. The other systems seem healthy.

A. P., aged thirteen, was admitted on account of loss of power in body generally. Up to six years of age, he seems to have been a strong, healthy child. When two years old, he is reported to have fallen from a high bed to the floor on his back, but according to the parents, this accident did not produce permanent injury. He began school at the age of five, but in the course of a year it was observed that he had difficulty in walking. He was easily tired, and tended to fall unaccountably. He was, therefore, removed from school, as the parents feared he might tumble into a canal along which he had to pass. His plump, healthy appearance remained undisturbed, but it was noticed that his gait became awkward, his legs being swung round, and his body swaying from side to side. Complaint was made of pain in the small of the back. The weakness gradually advanced, the tendency to fall increased, the muscles of the trunk and arms becoming affected, so that when down, he had difficulty in raising himself. The parents, who, as the history shows, have had ample reason to become observant, state that all this time they were of opinion that a number of the muscles gradually increased in size, while others diminished. Three years ago, the movements at the shoulder joint became much restricted. A year later, his legs absolutely refused to support the weight of his body, and he had to be constantly on a chair, bolstered up with pillows. His left arm was elevated by bending the head forward, taking the finger tips between his teeth, and raising the head again. Within the last year, the back has become much weaker, with a marked convexity posteriorly, and he had to be strapped to a chair. For a number of weeks he has been confined to bed.

Present condition.—Patient is a healthy-looking boy, with contented expression. His face and the backs of his hands have a faint brownish tinge, as if sunburnt. His facial expression suggests a constantly full mouth, and the large tongue tends to protrude, but there is no trickling of saliva. The head appears heavy, and inclines forward, but its movements are natural. He has a well-marked goitre. The patient usually lies or half sits in bed, his back supported by pillows, most of his weight resting on the left buttock. The left leg is flexed, and lies on its outer aspect, with the foot and ankle passed underneath the right knee. The right leg is also flexed, and lies on its inner aspect, the knee overlapping the left ankle. The right foot is partially extended, and lies partly on its inner edge, and partly on the inner half of the sole. The pose of the arms suggests

powerlessness, the upper arms lying closely applied to the side of the body, and the forearms resting on the thighs. The lower part of the thorax bulges considerably forwards, and a marked furrow exists between it and the full abdomen. From the third dorsal vertebra to the sacrum, the spinal column is curved backwards and to the right, the curve being greatest in the lowest part of the dorsal region. Patient cannot raise himself sufficiently to attempt standing, but he is still able slowly to bring his right hand to his head by the action of the trapezius and scapular muscles, and the muscles of the forearm attached to the lower end of the humerus. The left arm is quite helpless. Of the muscles, some are markedly increased in bulk, others reduced. The tongue is distinctly enlarged, and rolls about in a heavy, cumbersome fashion. The cheeks are relatively fuller and feel abnormally firm. This seems due to enlargement of the buccinators. The muscles of expression act sluggishly. The muscles forming the floor of the mouth are thickened. The latissimi dorsi are wasted. Both deltoids are full and firm in the lower half, but atrophied in the upper. The trapezius is wasted, especially of the left side. The inferior scapular muscles appear in fair condition. The right pectoral is atrophied, and the power is abolished, the left less so. Biceps is atrophied on both sides, brachialis anticus less so. Neither triceps is much altered. Supinators, pronators, flexors and extensors of forearm and hand are well represented, and power of movement seems natural, though sometimes there is loss of time in its initiation. The muscles of the hand appear intact. In the lower extremities, the calves are remarkably enlarged, and the buttocks to a less extent. Both sets of gluteal muscles are hard and firm, but there is almost total loss of power. The flexors and adductors of the thighs are firm, but almost powerless. The quadriceps extensor is hard and full, especially on the right, but power is much reduced. The hamstrings are more natural in appearance and strength. Both sets of calf muscles are much increased in bulk. There is a slight degree of talipes equino-varus. The muscles on the front of the leg are fuller and rounder than normally. Extension of the foot is possible, but power of flexion is lost. Of the back, the muscles on the right side appear fuller and firmer, those on the left are flabby. Both the atrophied and the hypertrophied muscles react less readily than normally to faradic and galvanic stimulation. No qualitative changes were detected. To the hand, the skin over the enlarged muscles feels considerably warmer than that of the rest of the body, but careful examination with the surface thermometer revealed no appreciable alteration. The organic reflexes seem fairly natural, though for the last few weeks there has been a

tendency to incontinence of urine. Patellar tendon-reflex is abolished, and there is no ankle-clonus. The superficial reflexes are natural. There are no sensory disturbances. There is no tenderness of the spinal cord. The intelligence is good, speech and memory are natural. In other respects, the boy seems in good health. He eats and sleeps well.

G. P., junior, aged eleven, was admitted on account of weakness in legs, and awkwardness of gait. He was in regular attendance at school up to the date of admission. There is no history of accident. Four years ago his parents observed that he walked more stiffly than was natural, and that his knees appeared weak. He used to trip occasionally. Two years later, it was remarked that the left foot was no longer placed flat on the ground in walking, the heel being distinctly raised.¹ This alteration grew more marked, and was followed in three or four months by similar appearances in the right foot. Both feet tended to drag, and stumbling became more frequent. He never complained of pain. He never showed the "waddling" gait of his brother, nor did he tend to fall down "all of a heap." His calves do not appear to the parents to have increased in size.

Present condition.—He is a strong-looking boy of 4 feet 9½ inches. Weight, 5st. 5½ lbs. Development and muscularity good. He runs about the ward, apparently with little difficulty. Close examination shows that he walks on his toes, his boots being accordingly altered in shape. This is most marked in the left foot. Both feet trip in walking, especially when the boy is tired. Both present the typical appearance of talipes equino-varus. The calf muscles are not enlarged, but feel firm. The tendo Achillis of both sides, of the left most markedly, is tense. The other muscles of the body seem natural. The organic reflexes are undisturbed. All the superficial reflexes are elicited with ease. The patellar tendon-reflexes and those of other tendons are exaggerated. Ankle-clonus is pronounced on both sides. Sensory functions are normal. The muscles of the left calf exhibit a diminished reaction both to faradic and galvanic stimulation. The muscles on the front of the same leg appear more excitable than is normal; and in the case of the tibialis anticus and long extensors of the toes, there is evidence of qualitative change. Bald patches, like those found on the father's, are present on the boy's head; and the dorsal aspect of the patient's toes are reddened, and present the appearance of

¹ The parents' observations are of considerable value, as long experience of disease had led them to watch the early development of symptoms with care.

chilblains. There is no cerebral disturbance. The spine is free of obvious deformity, and there is no tenderness or hyperæsthesia. The other systems are normal.

Remarks.—The cases are of interest as illustrating the occasional occurrence of different types of nervous disease in one family. That of G. P. (senior) is presumably one of gradually advancing spastic paralysis, probably excited by injury in a constitution predisposed to such affection through the previous ill-conditions of life. This view is supported by the history of the onset and the mode of advance,—the gradual increasing motor weakness, with rigidity of the lower extremities, and the comparative absence, from the beginning, of sensory disturbance. The exact significance of the sensory area, which apparently now exists at the lower part of the spinal column, is a question of some difficulty, but the presence of the area does not materially militate against the view that has been adopted. In addition to the spastic phenomena, his nervous system affords evidence of depreciation in the alopecic patches and other trophic disturbances. G. P. junior presents the early symptoms of a similar affection. The history and general characters, coupled with the exaggerated deep reflexes and ankle-clonus, are in favour of this view. The evidence of qualitative change in a single group of muscles was slight, and is insufficient to warrant dubiety with regard to the diagnosis, being, of course, capable of more than one explanation in harmony with this. Curiously enough, he, too, has bald patches on his head, closely resembling those seen in the father. A. P., on the other hand, is suffering from pseudo-hypertrophic paralysis, of which two older brothers have already died. He has no alopecic patches. The facts, therefore, suggest a transmitted tendency to primary lateral sclerosis, while the family history illustrates the known hereditary character of pseudo-hypertrophic paralysis; the former apparently passing in the direct line from father to son, the latter, as usual, through the mother to the male children only (see history). A further interesting question in heredity arises, in view of the fact that, of all the children, only the youngest son—born after the father's spastic symptoms had begun to develop—became affected in this special way, and this, apparently, in the absence of a direct, exciting cause.

In connection with the case of pseudo-hypertrophic paralysis, there are one or two other points of interest. The increased heat of the skin over the affected muscles speaks in favour of the view, that the seat of disturbance is the vaso-motor system. This receives important confirmation in the presence of the

well-marked goitre, without any indication of cretinism. Another curious fact is the presence of distinct pigmentation of the forehead and temples and backs of hands in a boy, who for months has practically been confined to the couch, recalling Dr. Gower's¹ case of pseudo-hypertrophic paralysis, seen along with Sir Wm. Jenner, where the skin of the temples, posterior axillary fold, abdomen, back and thighs, was so pigmented as to warrant the diagnosis of Addison's disease. The last point is the tendency to incontinence of urine, which, during the last few weeks, has begun to manifest itself—a late symptom to which reference has been made by Dr. Byrom Bramwell.²

¹ Gower's 'Pseudo-hypertrophic Paralysis,' p. 19, case 24.

² 'Lancet,' Aug. 9, 1879.

HYSTERO-CATALEPSY IN A MALE; ATTACKS SUSPENDED BY TESTICULAR PRESSURE.

BY ALLAN M'LANE-HAMILTON, M.D. (NEW YORK).

THE following case is of interest, not only by reason of its rarity, but because of the success that followed an interesting therapeutical experiment. To Dr. Robert Abbe, who kindly called me in consultation, I am indebted for the greater part of the following notes.

C. G. B., aged 35, married eight years. Father addicted to the excessive use of stimulants, and irascible. The patient is an intellectual man, and one of superior capacity. Mother of opposite type, a beautiful woman of quiet, even temperament, free from any trace of hysterical nature. Patient rather fat, and of one hundred and seventy pounds average weight. Health ordinarily excellent, though he has suffered from what was generally believed to be chronic peritonitis of several months' duration, eight months ago. He also had an attack five years ago, of pneumonia, with sequelæ, which one of his physicians thought was mild cerebro-spinal meningitis. Three years ago, while labouring under business reverses, he clandestinely began to take morphine—in eighth of a grain pills. The morphine habit grew rapidly until date, and he has never stopped it, and has taken at times as high as sixteen grains a day, but usually three grains in two doses, morning and night. His intellectual habit has been less bright during this time, though I have usually regarded him as lazy in mind, and from a boy disposed to exaggeration. His mother and senior brother (who is now in business with him) have for years been half credulous of spiritualism, but the patient has always scoffed at them. On the 16th of February last, he had a chill, which marked the beginning of pneumonia of a well-defined croupous type. He began convalescence after the tenth day, and was nursed by a female trained nurse, who resorted to massage quite frequently to quiet him from nervousness, supposed to be due to discontinuance of the morphine, which was unadvisable under the circumstances. He entirely gave up the drug rather abruptly during the fever, though some

frequent and considerable hypodermic injections were given for the relief of pain up to the crisis. On Monday, the 2nd of March, when convalescence was progressing, and he had sat up for two hours during the afternoon for the second time, he retired in a comfortable mood. The nurse had been dismissed, and he was alone with his wife. About nine in the evening he said he did not think he would sleep well. His wife tried to soothe him, but in a few minutes he began to show nervousness, and acted queerly. He began to kick off the bed-clothes, and act as if in a fit of petulance or temper; then turned over and beat the pillow violently, as if to vent his feelings. This was soon changed into a state of mental pre-occupation and moaning, quiet conversation in secret with imaginary people, exclamations of, "Oh, mother, mother!" as if she was seen in a dream. His fists were clenched and relaxed alternately; eyes rolled up imploringly, and apparently fixed on space. The head was occasionally buried back in pillow. Has general anæsthesia, though moaning, as if in a dream, "My head—my head; it aches so!" Pupils rather widely dilated, but reacting to candle-light rather sluggishly. He did not vary much from this state all night, except to remain quiescent for two or three consecutive hours, with apparent insensibility, and mostly complete unconsciousness. Towards morning the spell seemed to relax, and he half awoke, dazed, and declaring that the night had been a perfect blank, though sometimes he had seemed to answer questions intelligibly.

He drank milk freely, and seemed to have come to himself, but soon lapsed again into the queer unconsciousness. Many such relapses occurred during the day at intervals of half an hour, or at times two or three minutes only occurring.

They always began by a muscular fixation of the head backward on the pillow, eyes rolled upward a little, and lids open. (When I saw the case with Dr. Abbe, there was a slight tremor of the lids.) The ophthalmoscope showed a perfectly normal retina. The functions remained normal. Urine was passed in ordinary quantities, and the pulse never varied from 90, throughout the beginning, middle, or end of the attack, or the intervals. Temperature $99\frac{1}{2}$, not altered.

I saw him about twenty-four hours after the commencement of the attacks, and while sitting by his side he complained of headache, and after shuddering slightly, the fixation of the head just alluded to began to be apparent. There was first rolling upwards of the eyeballs, with a slight tendency to convergence, suspended respiration, and afterwards some slight oscillation of the eyeballs, and audible respiration. The colour of the face was, if anything, rather pale, and the lips were somewhat livid. When I extended the upper or

lower extremities, they remained in the position in which I had placed them, and there was slight, almost inappreciable, *flexibilitas cereæ*. There was no relaxation within a reasonable time—one or two minutes. With this condition there was very decided analgesia, and a pin was thrust into the surface, and the hair pulled, without any expression of suffering. The patellar tendon-reflex was, if anything, slightly exaggerated, though no cremasteric reflex could be evoked. In a period of about five minutes, there was some appearance of volitional return, for he opened his eyes, moaned, and placed his hands upon his head, and appeared to suffer. After rousing him, he was able to indistinctly call attention to his distress, but almost immediately he again became rigid. I this time extended and slightly adducted his hands and forearms, placing the tips of his little fingers and thumbs in contact, so that a sort of arch was presented. This implied a very delicate muscular co-ordination, and in a conscious, non-cataleptic person would require considerable effort. The position was maintained, however, without so much as a tremor, for two minutes. I then, bearing in mind the efficacy of ovarian pressure in corresponding states in woman, suggested to Dr. Abbe that he should make firm pressure upon one testicle: This he did, and almost *immediately* the rigidity relaxed, and the arms dropped. Coincident with this there was a return to consciousness.

Dr. Abbe, who closely watched the case, says, "Testicular pressure broke the charm, and they (the attacks) never returned." On the following evening he declared he was going to be nervous and sleepless again, and his wife said he began to act in the same way. I found him at 10 P.M. nervous, but apparently trying to control himself, yet kicking first one leg, and then the other, under the bed-clothes. He would lie quiet for a moment, and then snort and turn over.

I ordered his wife and nurse, who had returned, out of the room, and systematically bullied him for two hours, when he gradually quieted down, though not much sleep came.

During the catalepsy, his constant complaint on waking was that his head pained as if "*bursting*," front and back.

He had told me regarding sexual functions that, while vigorous, he had not had, he believed, more than two emissions during the act of coitus during two years past. The third day before the display above described was marked by three seminal emissions, without provocation or erection; they made him feel weak.

Others had occurred during the weeks he was in bed convalescing, and occasionally followed massage. The nurse had habitually taken temperature per rectum, and had noticed

an over-sensitiveness of the perineum, which made him squirm, and, on giving enemata, made her desist.

After the night of scolding, he rapidly changed, and not a nervous sign appeared again. He walked out on the fourth day after, and was well in a week.

This, so far as I know, is the first reported case of hysterocatalepsy in a male, though I believe that most of these cases of catalepsy met with among young subjects, of either sex, present an hysterical element.

A CASE OF MULTIPLE SIMULTANEOUS CEREBRAL HÆMORRHAGES, CAUSING HEMIPLEGIA AND OCULO-PUPILLARY SYMPTOMS.

BY W. HALE WHITE, M.D.,

Assistant Physician to Guy's Hospital.

JOHN R. B., æt. 44. Admitted into Guy's Hospital, Nov. 5th, noon. Eighteen months ago he had weakness in the left arm and leg, which came on slowly without any preliminary unconsciousness. He recovered his strength in about eight months, power returning first in the leg. On the morning of admission, whilst clipping a horse, the patient fell down insensible; he was not convulsed, but appeared "as if in a sleep." Within an hour he was brought to the hospital. He was then unconscious, and had passed his urine and fæces under him.

On Admission.—Face, pale. He lies on his back with extended thighs and legs, breathing stertorously and apparently in profound coma. Temp. 97·6; extremities cold; P. 56, hard and full; R. 32.

Nervous System.—The pupils are seen to be minutely contracted when the closed eyelids are raised; they do not react to light. Deviation of both eyes to left. Conjunctiva oedematous; corneæ sensitive to touch. No deviation of head. Jaws firmly closed. During inspiration alæ nasi dilate; during expiration the right cheek bulges more than the left, and the naso-labial furrow is less marked on the right side. The right arm and leg appear to be paralysed; there is slight rigidity of both. The thumb of the right hand is bent over into the palm and the fingers closed over it; when it is forcibly disengaged, it slowly returns to the same position. The left arm is freely moved about, and is occasionally convulsed, especially in the fingers and the flexors of the forearm.

Heart.—Obviously considerably hypertrophied.

Urinary System.—Urine pale; sp. gr. 1015, and contains a considerable amount of albumen.

1.45 P.M.—P. 72. The left eye is still deviated to the left; but the right eye was directed forwards. Pupils pin-point contraction, insensitive. Jaw relaxed and fallen. The right arm had been seen to move once or twice, as if involuntarily.

2.15 P.M.—P. 60; T. 97. Deviation of both eyes outwards; pupils still contracted.

3.15 P.M.—R. irregular, 24.

3.30 P.M.—Pupils dilated, but unequal; the right being much more dilated than left. Deviation outwards on both sides. Both arms are flaccid and mostly motionless, but both move occasionally.

4 P.M.—P. 95, weak and fluttering; T. 96.8.

4.30 P.M.—Death.

Post-mortem Examination.—Brain: all the three cerebral and the basilar arteries were thick and sodden-looking, no calcareous plates, or miliary aneurisms. The convolutions were much flattened. The left cerebral hemisphere was torn up by a large hæmorrhage, which had filled the ventricle with blood clot. The chief seat of the lesion was the internal capsule, and this together with the lenticular nucleus, external capsule and claustrum were completely destroyed. The antero-posterior extent of the injury was from the level of the anterior extremity of the corpus striatum back to the top of the posterior cornu. Externally, just the innermost part of the grey matter of the island of Reil was affected. Internally, the caudate nucleus was bulged over by the hæmorrhage into the right ventricle; the optic thalamus was destroyed, and it was apparently at the anterior part of this body that the intraventricular rupture had taken place. The septum lucidum was torn through; the right ventricle was full of blood, also the third and fourth ventricles, for some of the blood had passed down the aqueduct of Sylvius, which was full of clot; some blood had also passed out of the great transverse fissure to the base of the brain, and the clot was pressing on the left 6th, 7th, 8th, and 9th nerves, at their point of exit from the brain; the 7th, 8th, and 9th, on the right, were also pressed upon by clot, but only slightly. On section through the pons, a small recent hæmorrhage, about the size of a millet seed, was found exactly in the centre. Immediately under the centre of the floor of the aqueduct of Sylvius was an elongated hæmorrhage, which began at the extreme upper part of the aqueduct and extended nearly to its lower extremity. This elongated clot was about the thickness of a rather thick probe: round it were many punctiform excavations. In the descending motor fibres of the pons on the right side was a small old hæmorrhage, about the size of a pea, of a brownish colour. The kidneys were granular; the heart much hypertrophied, it weighed $26\frac{1}{2}$ oz. The testicles were fibroid, and there was an old hydatid cyst between the liver and the diaphragm.

The interest of this case speaks for itself. During life some difficulty was felt in accounting for the symptoms, and considering the condition of the pupils, those who first saw him placed the lesion in the pons; but it seems to me that the fact, that the hæmorrhage was sufficiently great to produce insensibility and considerable lowering of temperature, should have made them consider, that it was hardly likely that a hæmorrhage into the pons, sufficiently extensive to produce both these symptoms, should yet not affect the motor fibres going to both sides of the body. But the post-mortem examination explained these difficulties: for undoubtedly the first thing that happened was that a large hæmorrhage took place in the left cerebral hemisphere, and, bursting into the ventricles, caused, in addition to hemiplegia, insensibility and lowering of temperature; at the same time, the pontin hæmorrhage in the floor of the aqueduct of Sylvius caused the contraction of the pupils. The bleeding, probably, did not stop from either hæmorrhage during the patient's life, for the varying oculo-motor and pupillary symptoms can be explained easily by the pontin hæmorrhage affecting gradually different parts of the oculo-motor and pupillary centres in which it took place. The rigidity of the right arm and the occasional movements of both left and right are interesting also, as showing some irritation of the motor fibres. As when the patient was first seen there were no symptoms of the clot pressing on the left 6th, 7th, 8th, 9th nerves, it is probable that the blood did not reach them till later on, and perhaps did not clot till after death. The case has been recorded, because it is not very common to get, as in this case, three simultaneous hæmorrhages, viz. one ventricular and two pontin, although I have recently made a post-mortem examination in which severe meningeal hæmorrhage was associated with hæmorrhage into the pons. The possibility of more than one hæmorrhage occurring at the same time should always be borne in mind, if, as in this case, the grouping of symptoms is at all peculiar.

Reviews and Notices of Books.

A Text-book of Human Physiology, with Special Reference to Practical Medicine. By Prof. L. LANDOIS, of Greifswald. Translated from the Fourth German Edition, with Additions, by WM. STIRLING, M.D., Sc.D., Reg. Prof. Insts. of Med., University of Aberdeen. 2 vols. 8vo., with nearly 500 Illustrations. London: Griffin & Co., 1885.

THE first volume of Professor Stirling's translation of Landois' 'Human Physiology' appeared last year, and has been already noticed in these pages ('BRAIN,' vii. p. 534). With the publication of the second volume we have the complete work before us. The general impression left in the minds of most people by a general survey of its pages must, we think, be favourable, and the impression will certainly be strengthened by a more minute study of their contents. These are full and detailed, without being redundant; well arranged under distinct headings, and well indexed, so that any desired subject can be readily found without undue turning over of pages. The Text-book was already a good one in its mother tongue, it is in every way a better one in its English dress, and its title "Landois and Stirling" is quite justified by the amount of new matter which has been introduced by the translator. We cannot, however, help regretting the absence of references, which are so useful and prominent a feature in, e.g. Beaunis' 'Physiology': in a two-volume work such as the present, which is to be used by students of physiology as well as by students of medicine, the lack of references is a serious want. The labour of making these at all complete is, however, very great, and it would hardly be reasonable to expect to find them in a first edition: but we may hope to see the want supplied in a future edition of a book which is to take its place as a standard Text-book for advanced students. It is doubtless after due reflection, that Professor Stirling has added largely to the histological and anatomical departments by illustrations as well as by descriptions—additions which, however, have added to the bulk and price of the Text-book more than to its value as a compendium of modern physiology.

Of the 650 pages contained in the second volume, more than 450 are devoted to the Nervous System. Section X gives the Physiology of the Motor Apparatus; XI, General Physiology of the Nerves and Electro-Physiology; XII, Physiology of the Peripheral Nerves; XIII, Physiology of the Nerve-centres; XIV, the Sense-organs; and these, the most important chapters of

Physiology to the readers of this Journal, are also the most complete and satisfactory as regards treatment and arrangement. The instruments and methods used by physiologists in the investigation of the nervous system are very fully described, and the descriptions are made the more intelligible by diagrams and figures to which Dr. Stirling has made many valuable additions; the laboratory aspect of the subject is such as is useful and suggestive to those whose studies are chiefly clinical, while the clinical aspect is well kept in view, and cannot but be useful to those whose pursuits lie chiefly in the laboratory. It is further an advantage, that the exposition is encyclopædic rather than critical; the student who consults the work finds facts and statements to inform his own opinion rather than ready-made opinions. This feature is of no small value in a Text-book, and the only desideratum that remains is a reference to the original sources of the quoted facts and statements. As regards phraseology, we think that is a secondary matter in a Text-book like the present; it is, however, a relief not to meet with German-English in a translation from the German, and this is ample evidence of skilful interpretation that has cost much care. To the qualification of centres, as centres *for sneezing, for coughing, for vomiting, &c.*, exception might perhaps be taken as implying meanings rather more material than in reality, but for these Prof. Landois is responsible, and they are consecrated in most Text-books. On the whole we may feel assured that "Landois and Stirling" will be a leading text-book in this country, as "Landois" is already in Germany, and that it will exercise great influence in the teaching and in the study of physiology in the medical curriculum.

A. WALLER, M.D.

Recherches expérimentales sur le mécanisme de fonctionnement des Centres Psychomoteurs du Cerveau. Par le Dr. J. M. L. MARIQUE. 1 vol. large 8vo., pp. 140. Paris: Delahaye, 1885.

THIS little book is an aggregation Thesis presented to the University of Brussels. The first part is historical and critical; the second, experimental and critical. We need not say much here of the first, which contains a clear *exposé* of the various lines of argument adopted at different times and by different authors, in their attempts to prove or disprove the theory of cortical localisations. In the second, the author endeavours to establish the reflex "psycho-motor" mechanism of the cortex, partly by discussion, but chiefly by experimental methods, of which we shall endeavour to give a clear though succinct account. The point to be established is that the motor area is excited to action only by stimuli arriving to it from the cortical "sensorium commune," through

the fibres of associations which connect the different convolutions or lobes with one another.

Anatomical considerations show that it is possible, by means of rather superficial incisions, to isolate the sigmoid gyrus of the dog from the other cortical tracts of the brain, without injuring the peduncular and callosal fibres. For whereas the latter sink vertically into the white matter of the hemisphere, the fasciculi of association between the gyrus and the frontal parietal and temporal lobes, run near the surface and in a horizontal direction.

In order to avoid the disturbing effects of the anæmia of the motor gyrus which would necessarily attend division of the pia mater around it, the following method was adopted by Dr. Marique. A narrow blade is plunged to the depth of seven or eight millimetres at one of the angles of the gyrus, into the substance of the second frontal convolution, close to the motor area. The knife is then tilted, so that the point moves towards the median line, and a transverse incision is effected, dividing the grey matter and subjacent white matter as far as the corpus callosum, but leaving the pia mater and its vessels untouched. A second transverse incision is likewise made at the other angle of the gyrus; and a third, in a longitudinal direction, joins the inferior extremities of the two former.

In one series of experiments (ten dogs) the following plan was adopted. After the gyrus had been exposed, the animal was allowed to recover from the anæsthetic. Two hours afterwards it was etherised again, and the precise situation of the motor centres was determined by the induced current. The three incisions were then made as first described; and after the waking up of the dog, the effects produced were duly observed. Finally, a controlling experiment, to prove that the fibres connecting the cortex with the peduncle were intact, was made; the animal was etherised a third time and the induced current applied to the motor centres, when the characteristic movements on the opposite side of the body occurred.

The phenomena observed on the return of consciousness after the isolation of the motor tract by the three incisions are the same as those that follow the complete ablation of this tract. The animal first tries to bring into a natural position its extended limbs, but succeeds only in moving those on the same side as the lesion, and falls on trying to stand. After a short time it recovers the power of walking, but imperfectly, treading on the dorsal aspect of the carpus; and it cannot step over obstacles. Dogs accustomed to give their paws when ordered have lost the power of doing so with the parietic limb. The locomotor symptoms tend to disappear after the second day; but there remains for many weeks an inability to perform with the affected limbs action requiring a higher grade of co-ordination, or acquired by a process of training.

In a second series of experiments (five dogs) the three incisions were not made all at once, but singly, in order to determine the effects produced by each. The anterior transverse incision was

found to have but a very slight and transitory effect, nothing but a trace of paresis of the opposite limbs being observed. The inferior longitudinal incision produced a more pronounced weakness in those parts; whilst the posterior transverse cut was found to be almost as effectual as the complete isolation of the gyrus.

These results agree with what one would have expected, on the hypothesis of a reflex cortical mechanism. For, in the dog, the frontal region being but slightly developed as compared with the posterior convolutions, the share it takes in the psychomotor processes must be proportionally limited. The development of the temporo-sphenoidal region, on the other hand, places it in an intermediate position; and the experimental results, as we have seen, tally with the anatomical data.

Dr. Marique does not think that the symptoms of paralysis were in any way due to circulatory disturbances in the motor area, and he answers by anticipation this and other possible objections to the method he has employed. It is to be regretted, however, that he did not use the antiseptic method in his experiments. Still they appear to us sufficiently free from fallacies to establish his conclusions, which after all are but a confirmation of the view held by many, that the motor areas of the brain are to its sensorial tracts, what the anterior cornua are to the posterior in the spinal cord. We need not follow the author in his discussion of the question of substitution. He practically adopts the theory, that in the dog the lower centres are able to carry out the simple co-ordinated movements which make up the bulk of this animal's motor manifestations. The proof that the other half of the brain does not take up the functions of the injured hemisphere lies in this fact—that destruction of the former does not paralyse the limbs, the control of which it is supposed to have assumed. It seems to us also much more rational to assume that the corpus striatum, which in man and the higher apes may be considered as a mere surviving organ in the process of obliteration, occupies in the dog and other animals of the same grade an intermediate position: the cortical centres are only beginning to assume some of the functions of the lower centres which still retain a considerable potential power. We must not hastily assume, however, that because a dog deprived of his cortical centres reacts to ordinary stimuli in an apparently normal manner, it has not suffered loss in the psychological sphere, and that "substitution" can ever be complete. At any rate, much more complete observations than those we possess are required to establish this point.

A. DE WATTEVILLE.

Zehn Vorlesungen über den Bau der Nervösen Central-organe.

By Dr. LUDWIG EDINGER. (1 vol. 8vo., pp. 138, with 120 figs. Leipzig: F. C. W. Vogel, 1885.)

Scheme of the Functions of the Cerebral Nerves. By Professor JACOB HEIBERG, of Christiania. Wiesbaden: J. F. Bergmann, 1885.

THESE Lectures, delivered in 1884 to an audience of medical men, are distinguished chiefly by the numerous, and for the most part really admirable drawings of the various parts of the central nervous system, and illustrating both structure and naked-eye appearances. Many of the figures are diagrammatic, and if these are not always strictly accurate, they invariably express what they are meant to express, leaving no doubt on the subject. We do not know of similar detailed representations of cerebral structure in any single work. For the most part details are named on the figures; an immense economy of time, saving the annoyance of searching through a long letterpress description, to find perhaps that the structure sought is not named there after all, but in the text. It is not always possible thus to indicate structures on the figure itself, but there is no reason why it should not be oftener done than it is. The artistic work, in the great majority of these figures, is much superior to the woodcuts of our English textbooks.

The first Lecture is devoted to a short account of the history, and the methods of investigation, by which the structure of the central nervous system has been discovered. In other lectures there is no mention of authorities, and conflicting opinions are not as a rule stated. Lectures II. and III. give an excellent account of the gross anatomy of the brain, Dr. Edinger here as elsewhere making his figures serve in place of long word-descriptions. The remainder of the Lectures describe the structure and connections of the brain and spinal cord. Dr. Edinger explicitly states that he keeps as far as possible to received doctrines, without attempting to incorporate the results of his own observations on disputed points. Hence there is little ground for criticism. We need say, regarding the text, only that it is clear and uninvolved, and will be readily intelligible even to English readers. It should, we think, be specially useful to those who, wishing to take advantage of German work on the nervous system, are not always sure as to the identity of the structures named. In the text, Dr. Edinger gives both the Latin and the German nomenclature.

This is an ingenious attempt to represent, on two pages, the functions of the cerebral nerves. The method adopted is that of coloured type, motor nerves being indicated by red, sensory by yellowish-brown, and special nerves by blue. Compound nerves are indicated by the names being printed partly in each corresponding colour. The result is certainly striking, and conveys a large number of facts in small space. The student for whom it is

partly intended might, however, with the help of coloured pencils, make a synopsis which, although not so pretty, would be more useful to him than Professor Heiberg's ready-made one. The practitioner may with advantage use it as a reminder, but in doing so it will be well to remember, that there is more than a doubt as to the muscular supply of the palate muscles being derived from the facial, and also that the spinal accessory has other functions besides supplying the sterno-mastoid and trapezius muscles. The coloured printing and general execution of the synopsis is extremely good.

JAMES ANDERSON, M.D.

Report on Recent Advances in the Anatomy of the Nervous System. By JAMES ANDERSON, M.D.

WHILE our knowledge of the motor tracts of the brain and cord is far from complete, it is fairly definite and extensive. The same cannot be said of our knowledge of the sensory tracts, due partly to the greater inherent difficulty of the inquiry, partly to what may be called the accident, that the sensory fibres in the medulla oblongata and pons lie deeper than the motor.

Dr. E. C. SPITZKA makes a considerable contribution on this head in a series of articles on the Anatomy of the *Lemniscus*, with remarks on centripetal conducting tracts in the brain (*New York Med. Record*, Oct. 11th, 1884, *et seq.*). It will be remembered that the lemniscus appears as a flat triangular field, lying on the lateral aspect of the isthmus cerebri, stretching between the superficial origin of the fourth nerve above to the crusta below, overlapped in front by the transverse fibres from the posterior quadrigeminal body, and overlapping behind the anterior crus of the cerebellum. This, however, is only the exposure of a great tract which extends from above the pyramidal decussation through the interolivary region to the thalamus, gradually becoming more and more remote from the median raphé, while another detachment remains near it.

Professor Spitzka remarks, that while centrifugal fibres run clear from cortex to spinal cord, centripetal fibres are everywhere interrupted by ganglionic masses; an anatomical fact corresponding with the related physiological fact, that while the motor impulse is a comparatively simple affair, the co-ordination, for example, of space impressions from skin and ear into a general space sense is a complicated affair, requiring the intervention most probably of the cerebellum.

He quotes an important case published by him. "An elderly gentleman, after a small hæmorrhage in the pons, which caused unilateral ataxia of the opposite extremities, lived six years, during which time an intense secondary degeneration developed in the *descending* direction, and terminated after decussating in the opposite nuclei of the columns of Goll and Burdach, which were likewise atrophic." The columns of Goll and Burdach, at least

in their upper parts, degenerate centripetally, that is, towards their nuclei, so that apparently the two segments of this tract "degenerate toward an interpolated ganglion, justifying the assumption that functional impressions converge toward it, though not excluding the possibility of a continuous transmission brainward of sensory impressions at the same time." It will at once be seen that Professor Spitzka is justified in terming this suggestion a "startling innovation." The degeneration in this case descended through the interolivary tract of Flechsig, crossed in what Dr. Spitzka terms the *piniform decussation* in front of the hypoglossal nuclei (Meynert's fine upper pyramidal decussation), and so reached the degenerated nuclei of Goll and Burdach. Brown-Séquard found, that in spinal hemisection the muscular sense does not follow the rule of spinal decussation, but suffers on the same side as the motor function. Dr. Spitzka's case, along with others quoted by him, explains this apparent anomaly. The piniform decussation consists of fibres passing from the lenticular nucleus by the *ansa lenticularis* (*linsenkernschlinge*) which goes to form the main part of the lemniscus, and also a tract of undecussated centripetal fibres conveying muscular sense which probably ascends to the capsule independently, and so to the parietal lobe in close relation with the motor tract, a fact explaining the frequent disturbance of muscular sense present in hemiplegia. This latter, the lemnisco-pedal tract, or *Rinden-Schleife* of Monakow, degenerates downward only; the remainder, which is the main part of the lemniscus (formed by the olivary fasciculus and fibres from the piniform decussation), degenerates both upward and downward in different parts.

While acknowledging that to the atrophy and degeneration methods of Flechsig we owe the great advance during the last ten years in the anatomy of the brain and cord, Dr. Spitzka considers that certain of his assumptions are unjustifiable. For example, in his classical case of absence of the cerebellum, the cerebellum is far too important an element in the central mechanism to be eliminated without producing far-reaching defects of development elsewhere. Again, he states that from his observations he is becoming more and more convinced, that absolute reliance should not be placed upon the apparent continuity of myelinic development.

Dr. ALLEN STARR's Prize Essay on the Sensory Tract in the Central Nervous System (*Journal of Nervous and Mental Disease*, July 1884) contains not only an admirable summary of our present knowledge—and our present ignorance—on this subject, but also the detailed examination of the brain of a microcephalic infant, and much careful collation of cases. The infant lived for one week; it took nourishment, and cried like a normal infant, but made no voluntary movements, and seemed incapable of perceiving sensations. Medulla, pons, and cerebellum were present, but small. The prosencephalon was undeveloped, except a small detached mass between two layers of dura mater. The

thalamencephalon was represented by two grey masses lying anterior to the pons and cerebellum, from which arose the optic tracts and nerves, small in size. The other cranial nerves, from 3rd to 12th, were present. The pyramidal tracts were entirely absent. The interolivary tracts were partly developed, and showed the decussation of the fibres from the nuclei of Goll's and of Burdach's column. These fibres, the lemnisco-pedal tract of Spitzka, pass up in the external part of the lemniscus, and would appear, from their being present in this case, to develop from below upward, although, as we have just seen, Spitzka's case throws doubt upon this. The major part of the interolivary tract, *i.e.* the internal part of the lemniscus coming from the lenticular nucleus by the ansa lenticularis, was absent, developing apparently from above downward. Thus a continuous tract could be traced upward from the nuclei of Goll's and of Burdach's columns, across the median line through the interolivary tract into the lemniscus, and upward into the tegmentum of the crus cerebri, where it lay in the external lateral part of that body. The crustæ were entirely absent, as also the corpora quadrigemina. The red nucleus, and probably also the substantia nigra, were present. The formatio reticularis, the posterior longitudinal bundle, the nuclei in the floor of the fourth ventricle, the cerebellum and its peduncles, were normal. All the motor tracts, therefore, were absent; all the sensory tracts present. From this and two similar cases he concludes, that the sensory tracts must lie in the formatio reticularis in the lemniscus and interolivary tracts.

From this case and an analysis of other cases and observations, Dr. Starr draws the following general conclusions:—

1. In the cortex of the brain, sensations of touch, pain, temperature and the muscular sense are perceived.
2. These perceptions occur in the grey matter of the anterior and posterior central convolutions and of the parietal convolutions; sensations from one side of the body being perceived in the opposite half of the brain in a more intense degree than in the same half of the brain.
3. The various sensory areas for various parts of the body lie about and coincide to some extent with the various motor areas for similar parts—the area for the face, arm, and leg lying in the lower, middle, and upper thirds of the sensory-motor region respectively.
4. While the motor area is confined to the central convolutions, the sensory area includes, to some extent, the convolutions of the parietal lobe which lie adjacent and posterior to them. It is therefore more extensive than the motor area.

A.—The surface of the body is connected with a definite region of the surface of the brain by distinct tracts which convey sensory impressions. These impressions enter the spinal cord by the posterior nerve-roots, and then ascend in different tracts.

(a) The impressions destined to awaken the sensation of touch pass at once to the opposite half of the spinal cord, to a great

extent if not entirely, and ascend in the posterior white columns, the impressions from the legs passing in the posterior median, and those from the arms in the posterior lateral columns. On reaching the medulla these impressions pass to the *formatio reticularis* of the same side on which they were in the cord, and ascending in this tract through the pons and crus, reach the internal capsule, where they are conducted by a tract lying in the inner half of its posterior third to the corona radiata, whence they diverge to the cortex of the central and parietal regions, the impressions from the leg being perceived in the upper third of these regions, and those from the arm being perceived in the middle third of these regions.

(b) The impressions destined to awaken the sensations of pain and temperature also cross the median line immediately after entering the spinal cord, and pass up in the grey matter of the cord, probably in its posterior inner part. On reaching the medulla they enter the *formatio reticularis*, and from this point their course is identical with that of the impressions of touch already traced.

(c) The impressions destined to awaken the sensation of the location and of the motion of a limb ascend in the spinal cord, in the posterior white columns of the same side upon which they enter: the muscular sense from the legs passing up in the posterior median, and that from the arms in the posterior lateral column. On reaching the medulla, these impressions pass to the opposite side through the sensory decussation, then ascend in the interolivary tract to the pons, where they enter the lemniscus, and gradually turn outward from the median line as they pass up through the pons. In the crus, these impressions are conducted by the lateral lemniscus, which lies in the outer third of the tegmentum, and which passes directly into the internal capsule, there being situated in the middle part of the posterior third, external and in close approximation to the sensory tract of touch already described. From this point, the course of these kinds of impressions cannot be distinguished from one another, and their termination is in the cortex of the central and parietal regions, the muscular sense of the leg being perceived in the upper third, and that of the arm in the middle third, of those regions.

B.—Sensory impressions from the face enter the pons by the sensory root of the trigeminus, and pass downward to sensory cells which lie in the lateral portion of the *formatio reticularis*, and which are ranged in a column extending from the junction of the upper and middle third of the pons, to the lower limit of the medulla. The upper portion of this column receives the fibres from the upper branch of the nerve; the middle portion from the middle branch, and the lower portion from the lower branch. The course of sensory impulses, from these sensory cells to the brain, is in the lateral part of the *formatio reticularis* of the same side upon which they enter, up to the junction of the upper and middle thirds of the pons, where they cross the median line and join the sensory impressions from the body in the *formatio reticularis*. In the crus cerebri, these impressions pass in the outer half of the tegmentum, and, thence entering the posterior third of the internal capsule,

pass on to the corona radiata. In the divergence of fibres in the corona, these impressions pass along the lowest radiation, and thus reach the lower third of the central and parietal regions, where they are perceived. It is, as yet, impossible to distinguish between the course of tactile, painful, and muscular impressions from the face to the cortex of the brain.

C.—Sensory impressions from the great cavities of the body enter the spinal cord by the posterior nerve-roots, and are probably received by sensory cells which lie in the inner and posterior part of the grey matter of the spinal cord—the Clarke column of cells. From these cells, these impressions pass outward to a white column lying in the lateral periphery of the cord—the direct cerebellar column—in which they pass upward to the medulla, and on through the restiform body to the cerebellum, to terminate either in the nucleus dentatus, or in the cortex, or in both. The existence of a decussation of these impulses is undetermined, but, if it occurs, it must take place soon after their entrance, as each direct cerebellar column passes to that hemisphere of the cerebellum of the same side upon which it lies. The impressions thus transmitted to the cerebellum are not those of touch, pain, temperature, or the muscular sense, and are probably impressions connected with the functions of vegetative life and destined to awaken reflex actions.

In a recent issue we discussed the first two of Professor BURT WILDER'S "Cartwright Lectures on the Methods of Studying the Brain." The third Lecture (*New York Med. Journ.*, April 5th, 1884, *et seq.*) is divided into two parts; the first devoted to the completion, correction and general castigation of our anatomical textbooks, and chiefly of the two which Professor Wilder tersely denominates "Quain" and "Gray;" the second, to a discussion of the methods of illustration and description, with a revision of encephalic nomenclature. Professor Wilder considers that we place too implicit a trust in the two works alluded to, and treats of their omissions, inaccuracies, and ambiguities.

As to omissions, we may at once set at rest the anxieties of any junior readers by saying, that those pointed out by Dr. Wilder are neither very numerous nor very important. We scarcely think that the editor of 'Gray's Anatomy' would claim for it completeness in every detail. The omission from it, for example, of a description of the tractus transversus pedunculi of Gudden, rechristened the Cimbria by Dr. Wilder, can scarcely be said seriously to impair its value as a student's textbook of anatomy. The same omission even in 'Quain's Anatomy' cannot be regarded as a grievous sin, although doubtless it ought to have been mentioned, seeing that that work, at least in this part of it, is no longer a student's textbook, but a work of reference. The account of the nervous system in 'Quain's Anatomy' is well and copiously illustrated, but Dr. Wilder reproaches it for not containing diagrams of the bulbus cornu posterioris and of the cauda striati, both described in the text. Dr. Wilder considers that the striæ longitudinales callosi are merely the prints of the anterior cerebral

arteries; but opinions may very well differ on this matter, and the absence of reference to this cannot be characterised as an omission. This completes the list, and its shortness is surely a strong testimony in favour of our textbooks. The inaccuracies, Dr. Wilder admits, are "comparatively trivial." He first shows elaborately that the *centrum ovale majus* does not exist, because the upper surface of the corpus callosum is concave transversely, and convex antero-posteriorly, so that a horizontal section on the level of the corpus callosum would open the lateral ventricles. This simply means, that the section to expose the broad white surface called *centrum ovale majus* is not a plane surface, but a doubly curved one. The complete circumscription of the end of the descending horn of the lateral ventricle is implied in the usual accounts, but is certainly not definitely stated as it ought to be. A more important correction, however, is contained in Dr. Wilder's description of the *commissura fornicis*, the thin lamina which unites the two thicker lateral masses of the fornix. This lamina is a secondary formation, the two halves of the fornix being originally distinct, a fundamental fact, the recognition of which is necessary to an intelligent conception of cerebral architecture.

For seven years, Dr. Wilder states, he has been in the habit of beginning the account of the brain, whether for anatomical or physiological purposes, by a description of the cavities, their mode of origin, their succession, &c., the parietes being described as built up around them, and gradually differentiated. There is much to recommend this method, and we fully agree with Dr. Wilder that, both in the figures and in the text of our standard anatomical works, there is much indefiniteness on the details of what he terms "coelian circumscription," that is, as to the boundaries of the ventricular cavities. We cannot share Dr. Wilder's scepticism as to the existence of the Foramen of Magendie. Difficult as it is to believe in its existence *à priori*, there can be no doubt whatever of the fact, explain it as we may. To the Foramen of Monro Dr. Wilder does full justice, and to any one who really wishes to understand the part of the cerebrum usually known as the transverse fissure, the relation to this of the *velum interpositum*, and the precise formation of the Foramen of Monro, we recommend a careful perusal of Dr. Wilder's description. He insists also on the fact, now well recognised, that the "fifth ventricle" is no part of the true ventricular cavity, has in the adult no communication with it, and never had any, but is merely a part of the longitudinal fissure separated off by the downward curvature of the corpus callosum.

Dr. Wilder makes some excellent suggestions regarding figures which he would use not merely to supplement, but, as far as possible, to replace description. He would have them made more numerous and more definite, and he suggests rules for the placing of them in order to facilitate easy reference and comparison. Some of these rules we should have transcribed, but that Dr. Wilder, as already stated, writes in an unknown tongue, *e.g.* he tells us that "transections of a symmesal object should form a horizontal

syntropic series." The general object is to secure uniformity, and thereby save the labour involved in mentally reversing figures. One recommendation we should very gladly see generally adopted, namely, that the full technical names of parts should as far as possible be given on the figure, or by the side of it, with indicator lines. The economy of time by the general adoption of this plan would be very great. The last part of the third Lecture, devoted to a revision of nomenclature, was not delivered, but the substance of it is given in the *New York Med. Journ.*, March 21st and 28th, 1885, to which we must refer the reader.

Abstracts of British and Foreign Journals.

Fol on the Micrococcus of Hydrophobia.—At the meeting of the Académie des Sciences held on the 14th of December, 1885, Professor H. Fol, of Geneva, stated that after many endeavours he has found a method to demonstrate in the spinal cord of hydrophobic animals, certain bodies which are not to be seen in the healthy tissues. The microscopic sections must not be thicker than $\cdot 02$ of a millimetre, and the hardening and staining processes carried out according to the methods of Ehrlich and Weigert.

Under such conditions, and in specimens otherwise perfect, groups of rounded bodies—micrococci in all probability—are seen to be lodged either in the lamellæ of neuroglia, or sometimes in the annular spaces comprised between the cylinders (stained dark blue by the hæmatoxylin) and the sheath of Schwann (buff coloured). Occasionally they are found in certain cavities, the size of a myelin fibre, of which the histological nature is unknown.

The globular bodies are perfectly spherical, defined, and stained dark violet; they usually lie scattered, not in strings; though at times are found to form figures of 8, showing a scissiparous mode of multiplication. Their average diameter is $\cdot 0002$ millimetre.

An appropriate medium, sown with rabid brain-matter, and kept at a certain temperature, shows a slight turbidity which on the fourth day falls to the bottom of the liquid. This deposit has the power of giving hydrophobia to animals inoculated with it, though the period of incubation is longer than when the original matter is used. The same deposit applied to a cover-glass, dried and treated with the bichromate and copper solution, then coloured and uncoloured in the same way as the cord, shows the same groups of micrococci, stained dark violet. Inoculation of cultures of more than six days old does not produce any marked symptoms. It would be interesting to ascertain whether this is due to an alteration of the virus, and whether animals so treated become proof to the poison.

Horsley and Schäfer on the Muscular Contractions which are evoked by Excitation of the Motor Tract.—At a meeting of the Royal Society, held on the 10th of December, 1885, the authors described the results of a large number of experiments undertaken, in order to determine the character of the muscular contractions which result from excitation of the several parts of the motor tract, especially with reference to the rhythm with which the skeletal muscles respond to such excitation.

For the purpose of their experiments they considered the motor tract under four heads, viz.:—1. Its commencement in the nerve-cells of the cerebral cortex. 2. The connection of these cells with the lower nerve-centres by the nerve-fibres in the corona radiata. 3. Its continuation along the medulla oblongata and medulla spinalis (including the nerve-cells of those structures). 4. Its peripheral continuation along the motor nerves.

Their method of proceeding was to excite these several parts in succession, and record the contractions of one of the limb muscles upon a moving blackened surface, either by directly connecting the tendon with the lever of a myograph, or by Marey's method of transmission by tambours and india-rubber tubing, the time being simultaneously recorded upon the moving surface by a clock marking seconds. Usually the rate and duration of the excitation were also recorded by a small electro-magnet. Besides the contractions resulting from electrical excitation, they frequently obtained an accidental record upon the moving surface of spontaneous or voluntary contractions of the muscle, the responses of which to electrical excitation of the cortex cerebri they were preparing to record, and they have thus been able to compare these records of voluntary contractions in animals both with the results of electrical excitation of the several parts of the motor tract in the same animals, and with records of voluntary contractions in the human subject. They also studied, in the same way, the epileptoid contractions which are often found to follow a period of electrical excitation of the cortex cerebri in animals, and compared these epileptoid contractions with numerous others recorded by one of them from cases of true epilepsy and other affections of the nervous system (in man and animals), accompanied by rhythmic muscular movements.

Results.—It has generally been admitted by authors, that so far as regards the rhythm of muscular response, the result of exciting either the cerebral cortex or any other part of the motor tract is precisely the same as that which is well known to be the case with

the excitation of the motor nerve, namely, that for all rates of excitation the rhythm of muscular response is identical with the rhythm of excitation. The experiments of Horsley and Schäfer show, on the contrary, that this statement only holds good for low rates of excitation up to about ten or twelve per second, but that for all higher rates of excitation of the cortex cerebri, corona radiata, or medulla spinalis, the muscular response does not vary with the rate of excitation, but maintains a constant rhythm, which is independent of the excitation rate and approximate to ten per second.

The muscle curves which obtained from different mammals as the result of successive excitation of the cortex cerebri, corona radiata after removal of the superjacent cortex, and of the cervical cord after section below the medulla oblongata, are very similar to one another, and exhibit along their course, both at the commencement and during the whole extent of contraction of the muscle, small but distinct undulations following one another at the rate of about ten per second, with very considerable regularity, although in a few instances the rhythm may be a little slower or faster than these (eight to thirteen per second are the extreme variations observed). These undulations have the same rhythm and character whatever the rate of excitation (unless this be allowed to fall below about ten per second). Moreover, precisely similar undulations are always visible upon the myographic curve of all voluntary or spontaneous contractions (including reflex contractions) both in the lower animals and in man.

It is further noted, that in the record of the contractions of epilepsy there can frequently be seen marked upon the larger curves, produced by the relatively slow clonic spasms, smaller undulations succeeding one another with a rhythm of eight or ten per second. In some cases the clonic contractions themselves may attain this rate, but they are then always simple and without any indications of smaller waves.

In a very few instances out of a very large number of experiments, there occurred upon the tracings obtained as the result of rapid excitation of the cortex cerebri, corona radiata, and medulla spinalis, besides the usual well-marked undulations of the rate of about ten per second, other very minute waves upon these undulations, corresponding in rhythm with the rate of excitation. These were the only occasions in which we have obtained results at all similar to those mentioned by Franck and Pitres.

The authors showed tracings indicating the general nature of

their results, from which they draw the following and main conclusions: 1. That the normal rate of discharge of nervous impulses from the motor nerve-cells of the spinal cord along the motor nerve-fibres is approximately 10 per second. (The rhythm of a clonus (*e.g.* ankle-clonus) is also about 8 or 10 per second, and that of strychnine tetanus in the frog, as indicated by the electrical variations of the muscle current, has also about the same rate (according to Lovén). 2. That in the case of nervous impulses reaching these nerve-cells in more rapid succession than about 10 per second, a process of summation occurs within those nerve-cells, so that the rate of discharge remains about the same in all cases. 3. That the nervous impulses which produce a voluntary contraction also traverse the motor nerve-fibres at about the same rate. There is, however, no evidence to show whether this rhythm of the volitional impulses is generated in the cells of the cerebral cortex, or in the cells of the lower nerve-centres. 4. That the slower rhythm which is often exhibited in epileptoid contractions is the result of a further summation, but there is no distinct evidence to show where this occurs. 5. That occasionally, though rarely, the summation of rapidly succeeding nervous impulses may be only incompletely effected within the nerve-cells of the spinal cord, or may not occur at all. In these cases results similar to those of Franck and Pitres are obtained.

Hale White on the Pathological Histology of the Semilunar and Superior Cervical Sympathetic Ganglia (*Trans. Roy. Med. Chirurg. Soc.* 1885).—This paper is founded on an examination of about a hundred and fifty sections of the above ganglia, from which it is concluded that the external appearances of the ganglia are of no importance whatever in pathology, unless they be obviously affected by malignant disease, abscess, tubercle, &c., or have become implicated in some inflammatory condition of surrounding parts. The typical ganglionic nerve-cell resembles other ganglion cells; it is larger and more rounded than those from the anterior cornua. No inference can be drawn from the number of nerve-cells to be seen in a section, for the normal arrangement is irregular, the sections are not in the same plane, and all the ganglia are not the same size. Many cells have no processes, and some have neither nucleus nor nucleolus. As a general rule, the cells are wasted in patients who have died of wasting diseases; but this is subject to many exceptions. Pigmentary degeneration may or may not be accompanied by a diminution

in the size of the cell, and is almost universal in the sympathetic ganglia; perhaps this may be the cause of some of the slight ailments from which we all suffer. In the present state of our knowledge, the varying appearances from pigmentation have no significance. Pigmentation of nerve fibres does not occur. The amount of fibrous tissue varies very much in different specimens, and in different parts of the same specimen. The superior cervical ganglion contains more blood-vessels than the semilunar; in the latter there is no constant arrangement. In chronic Bright's disease the small arteries are thickened; otherwise in this malady the specimens are normal. In acute inflammation of the ganglia the section is obscured by the enormous number of escaped white blood cells, there is also multiplication of the connective tissue-cells. This may be accompanied by congestion. It has been found only in diabetes and purpura hæmorrhagica, and is the only pathological lesion about which one can speak with certainty.

A. DE WATTEVILLE.

Fournier on the Mental Disorders of the Præataxic Stage of Syphilitic Locomotor Ataxy (*L'Encéphale*, Nov. 1884). Extract from a series of Lectures on the Præataxic Stage of Syphilitic Tabes.—Mental disorder, the Author states, may occur in various stages of Locomotor Ataxy.

(I.) It may, in some exceptional cases, mark the very commencement of the disease. In one patient, who eventually suffered from well-marked tabes, that disorder was ushered in by the following mental symptoms: dulness and blunting of the intellect; slowness of ideation, inaptitude for any occupation requiring close attention; rapid cerebral fatigue caused by the least work; in a word, intellectual torpor, intellectual asthenia. This condition lasted about a year, and then, singularly enough, entirely disappeared, apparently yielding to antisyphilitic treatment.

Another patient of this class, before becoming ataxic, became incapable of following his usual employment; without being incoherent he had curious bizarre ideas; he was sad, melancholy, depressed, and during several months was pursued by thoughts of suicide. After awhile all these symptoms passed away, to be succeeded, however, by others of the usual tabetic character.

From the above it would appear, that in the præataxic stage of tabes we may meet with transient mental affections, just as at about the same time, certain forms of paralysis occur which are of equally short duration. Thus the paralysis of ocular muscles, so

common a forerunner of ataxy, may have as a pendant certain forms of paralysis or paresis, so to speak, of the intellect, equally destined to disappear with more or less rapidity. The author thinks that probably these two orders of symptoms have a similar origin.

Occasionally the mental disorder, appearing at the very commencement of the præataxic stage, may be permanent, the tabes being then inaugurated by symptoms resembling those of ordinary general paralysis, which the author has elsewhere described under the name of "syphilitic general pseudo-paralysis."

(II.) It is much more common, however, for these psychical phenomena to appear at a time when the præataxic stage has made some progress. That is to say, they do not usher in the disease, except in the rare cases mentioned above; but they may be the first symptoms of a cerebral character, being themselves followed by others, such as vertigo, congestive attacks, aphasia, convulsions, hemiplegia, &c.

Clinically in one group of cases the symptoms are as follows:—weakening of the memory, modification of character, of temper, of the habits; an intellectual asthenia slowly progressing towards a final condition of hebetude. Of all the intellectual faculties the memory is that which, in most cases, is soonest and most severely affected, and this peculiarity, it may be remarked, resembles what occurs in cerebral syphilis, in which disorders of memory are common, and frequently severe.

This enfeebling of the memory in most cases progresses slowly; but its onset may be sudden, occurring after a congestive attack, or, what is more usual, after an epileptic crisis. Sometimes it even appears to increase by sudden gradations, each epileptic seizure or congestive attack leaving an increased weakness of memory, and making a notable stage in the progress of the amnesia.

Finally, in the præataxic stage we may, in some rare cases, meet with transient attacks of amnesia lasting from twenty minutes to half an hour, the memory then remaining for a time uncertain and faulty, but gradually recovering.

In another group of cases, temper, character, daily habits, are modified; and this is what enquiry into such a case reveals:—the patient has undergone a sudden moral transformation; has become reserved, gloomy and morose; he then loses his spirits and his vivacity, caring for nothing—either work or pleasure. He becomes idle and indifferent, inert and apathetic, and even ceases to care for himself. Now and then suddenly his dulled vitality wakes in the form of impatience, anger, and motiveless passion.

Or, in a third class, the intelligence becomes diminished, just as in cerebral syphilis; there is a species of intellectual asthenia, a slowness of ideation; conception of ideas is less facile, less clear, and less precise; aptitude for mental work, for business, for every occupation requiring an association of ideas is diminished; brain-weariness is brought on, not only by real intense thought, but by ordinary reading, by prolonged conversation, or even by listening to a stage play. As time goes on, the mental disorder increases, and the patient becomes incoherent and truly insane, falling into a condition of incoherent hebetude, this being but a stage in a disease which ends in that abolition of the mental faculties called dementia. It is rare, however, for the mental degeneration to progress thus far before the appearance of the usual symptoms of motor inco-ordination.

Twice the author has seen *Tabes* ushered in by a series of cerebral symptoms, more complex, and almost identical with those of General Paralysis of the Insane, to which he gives the name of "Syphilitic pseudo-paralysis." The symptoms in cases of this kind are as follows:—(1) Loss of memory. (2) Stammering, hesitation, slowness in delivery of words, a sing-song speech, omission of syllables in words. (3) Mental disorders, such as dulness, slowness, and stupidity, followed by more marked signs of mental deterioration. The patient is said to be "wrong in the head." He is unfit for business, is forgetful, makes mistakes, and after a while enters into another phase. He then shows great hebetude, but has none of those delusions of grandeur so common in ordinary general paralysis; he is rather in a happy state of torpor, giving way frequently to motiveless laughter, all desire and all will being lost. Then come on motor troubles, clumsiness, and muscular weakness, with a slow, hesitating, uncertain gait, inequality of pupils, difficulty and hesitation in articulation, tremor of the lips and of the facial muscles when the patient tries to speak. After a while he becomes incapable of uttering a complete sentence, an idea being only expressed by a few words slowly and painfully uttered; but still a certain degree of consciousness and even of lucidity is preserved, and the patient manages to answer a few questions and execute a few voluntary movements. In a later stage the pupil is immobile; there are amblyopia and amaurosis, incontinence of urine, abolition of the reflexes, with undeniable ataxy, which is, however, less marked than in ordinary cases of *tabes*. After long vegetating in this miserable state, the patient enters the final stage of his malady, his mental faculties becoming

gradually extinct. At the autopsy we find general thickening of the membranes, and especially of the pia mater, with lesions of the grey substance of the convolutions and enormous dilatation of the ventricles. In the cord we find the lesions characteristic of tabes.

Here, the author thinks, we have either a cerebral syphilis with consecutive tabes, or tabes commencing in the cerebral form, the latter being, in his opinion, the more probable; and he considers the malady remarkable in that it assumes the form of general paralysis, or, to be more exact, the form to which he gives the name "pseudo-paralysis" of syphilis. He is thus of opinion, that syphilitic tabes may be initiated by a series of cerebral symptoms, closely resembling those of ordinary general paralysis.

On Disorders of Sensation in Cerebral Hemiplegia. By Dr. LEGROUX and Dr. DE BRUN (*L'Encéphale*, 1884).—The disorders of sensation met with in hemiplegia are here classified into five groups:—

(1.) Sensation is unaffected, the paralysis being purely motor.

(2.) The anæsthesia affects the whole surface of the paralysed members.

(3.) The anæsthesia is absolute in one extreme segment (thigh or foot, for example), whilst it is much slighter in the middle segment, and still less in the other extreme segment. Thus, the foot may be absolutely anæsthetic, the leg less so, and the thigh almost unaffected. These difficult degrees of sensibility are not sharply defined, but fade gradually from one into another.

(4.) The external half of a limb has its sensibility normal, or slightly enfeebled, whilst the internal half is almost absolutely anæsthetic.

(5.) The anæsthesia is in patches of irregular distinction, zones of absolute anæsthesia being surrounded by surface but slightly affected; or we may find patches on an anæsthetic limb where touch remains almost normal. In this variety the patches of altered sensibility have sharply defined margins, and instead of gradually passing away, the anæsthetic zones may shift about from day to day; a patch yesterday anæsthetic may to-day be normal.

The explanation of this oscillation of the anæsthesia is difficult; but since the cerebral lesion does not shift, it is concluded that the sensory disorders of hemiplegia do not obey the same laws as the motor affections, and do not correspond to fixed localised lesions of

the cortex. These disorders of sensation present other particulars, in which they differ from the motor affections. With respect to the mode of onset, for instance, we may often see the motor paralysis increase for three or four days before arriving at its maximum of intensity, while the anæsthesia attains its height at the very outset; or the motor disturbance increases whilst the anæsthesia is diminishing.

Many writers have shown the possibility of cure in these cases of anæsthesia by methods such as metallotherapy, usually applied in the sensory affections of hysteria. The authors then show how, if small patches on these anæsthetic limbs be rapidly pricked with a needle, the sensation will return, and this even in cases of long-standing hemiplegia with contraction.

The anæsthesia of cerebral hemiplegia presents many points of resemblance to that of hysterical, syphilitic, or toxic origin, showing that it is due to some alteration in the blood, or to a sudden or gradual modification in the pressure in the cerebral vessels. The oscillations, fluctuations, and variability of the anæsthesia can only be explained by cerebral phenomena, which, though tangible enough, are certainly transient. This view is supported by a consideration of the sensory phenomena so often premonitory of hemiplegia due to atheromatous vessels, which, closely resembling those under consideration, is caused by an alteration in the cerebral circulation. Again, the anæsthesia seen at the outset of general paralysis, is due to a congestion of the cortex preceding the chronic encephalitis.

Thus it seems extremely probable, that the disorders of sensation in question are due, in many cases at least, to an alteration of the cerebral blood-pressure. Of course it will be understood, that those cases in which the anæsthesia is caused by a lesion situate in the posterior part of the external capsule are here excluded from consideration.

On the Slaty Discoloration of the Brain in General Paralysis and its connection with Bedsores. By Dr. BAILLARGER (*Annales*, 1884).—This paper treats of a discoloration of the brain met with in hues varying from slate to almost black, and sometimes described by the term greenish, most commonly observed at the autopsies of general paralytics who have suffered from bedsores.

As a rule, the author finds this discoloration confined entirely to the cortical grey matter, being sharply defined by the junction

of the cortical with the white substance; but now and then it is observed in the basal ganglia.

The tissue thus affected is generally, but not invariably, softer than the surrounding convolutions, and is at times in a state of ramollissement. The base of the brain and the cerebellum are the parts most commonly thus affected, but the surface of the hemispheres does not always escape. The microscope reveals a granular stroma, with scattered nerve filaments and numerous cholesterine crystals. According to Calmeil, the discoloration is due to the infiltration of the affected tissue by numerous pus-corpuscles.

A foetid odour is noted in about half the cases, but may be absent even when the softening is well marked.

The pia mater and arachnoid may present only the changes of general paralysis; but more commonly there is a purulent infiltration of the pia, at times completely hiding the convolutions. The arachnoid cavity may be empty, but generally contains a varying quantity of flaky serosity, a foetid odour being occasionally present. Changes similar to these are met with in the cord and its membranes, with perhaps even greater frequency than in the brain.

These appearances are most commonly found at the autopsies of general paralytics, presenting large and deep sacral bedsores, and are due in many cases to an ascending cerebro-spinal meningitis; but it is important to notice that the discoloration in question has been met with when the meningitis was absent.

The author divides the cases into two classes:—

(1.) Those in which we find cerebro-spinal meningitis with infiltration of the pia mater with an ichorous pus. Here the discoloration is probably due to a simple imbibition of pus by the subjacent grey matter.

(2.) Those in which the discoloration is observed without the meningitis. In such cases the explanation above given will not hold, but the change is attributed to an inflammation of the affected tissues, due to the deposit in them of ichorous pus absorbed from the bed sore by the blood-vessels, and characterised especially by the putrid odour and tendency to gangrenous degeneration.

That we most commonly find this lesion in the brains of general paralytics is readily explained by the relative frequency of bedsores in the later stages of that affection.

Now and then a purulent spinal meningitis is set up by a bed-sore, without being accompanied by any discoloration of the brain.

R. ATKINSON, F.R.C.S.

Westphal on the Patellar Reflex in Diagnosis. WESTPHAL (*Archiv f. Psych.* Bd. xv. p. 731) was consulted in 1878 by a man aged 45, who had exhibited secondary symptoms of syphilis four years before. He was hypochondriacal, and extremely nervous and anxious about himself, and had recently had a transient attack of hemianopsia ("he saw the half of things"), and amnesia. There was an absence of all symptoms of nervous disease, with the solitary exception, that the patellar reflex could not be evoked. This, however, was proof to Westphal of an affection of the external parts of the posterior columns of the cord in its upper lumbar portion, or at the junction of the lumbar and dorsal portions. Two years later there was commencing optic atrophy in both eyes, which in another two years had progressed to absolute blindness. And then, suddenly, an attack of acute mania with grandiose ideas, and slight impairment of speech developed, and the patient died. During the whole course of the case there were no symptoms of ataxia, or indeed of any affection of gait, and there were no noteworthy pains; there was, however, at times, some difficulty in emptying the bladder. The *post-mortem* confirmed the diagnosis. There was sclerosis of the posterior columns throughout the cord, the disease being most extensive in the lower part of the dorsal division of the cord. Westphal discusses the etiology of these cases. He thinks that the syphilitic taint is only of secondary importance; the prime factor is an inborn psychological tendency, a peculiar disposition of the elements of the brain and spinal cord.

Remak on the Tendon-Reflexes and the Reaction of Degeneration. REMAK (*Archiv f. Psych.* Bd. xvi. p. 240) has studied the relationship that exists between the tendon-reflexes and the reaction of degeneration, with a view to aiding the differential diagnosis of amyotrophic paralysis of spinal, and peripheral origin, respectively. He summarises his results in the following conclusions:—(1) Exaggeration of the tendon-reflexes (more especially the phenomenon of ankle-clonus), with partial reaction of degeneration, has only been observed in cases of amyotrophic lateral sclerosis. (2) A normal condition of the tendon-reflexes with well-marked partial reaction of degeneration has only been observed in atrophic spinal paralysis, poliomyelitis anterior. (3) Absence of the tendon-reflex occurs, (*a*) with abolished excitability of the nerves, in all severe cases of amyotrophic flaccid paralysis, whether of spinal (poliomyelitic) or peripheral (neuritic) origin—and where there is resolution, the tendon-reflexes are still absent long after the

galvano-muscular reaction of degeneration has disappeared; (b) in the lighter cases of primary peripheral degenerative neuritis of mixed nerves, in which perhaps there is no paralysis; (c) in cases of absolute peripheral paralysis (e.g. from compression), even though there is no reaction of degeneration.

Sakaky on Degeneration of Peripheral Nerves in Tabes Dorsalis. WESTPHAL, seven years ago, examined the cutaneous nerves of the lower extremities in a case of tabes, and discovered that the number of double-contoured nerve-fibres was greatly lower than in normal nerves. Déjérine, three years ago, observed similar changes in two cases, which he described as peripheral neuritis, and regarded as a parenchymatous affection. The same author has recently reported two cases which presented the symptoms of tabes, but which showed no disease of the spinal cord, though the cutaneous nerves were atrophied. Sakaky (*Archiv f. Psych.* Bd. xv. p. 584) gives the result of a very careful examination he made of the nerves in a case of tabes. His observations show that in tabes there may be atrophy of the peripheral nerves, especially of the cutaneous branches. He is unable to state where the atrophy begins; in some nerves the peripheral portion was diseased, in others the central portion. He thinks that the presence of anæsthesia may perhaps be an indication of the atrophy of a nerve.

Kirchhoff on the Localisation of the Ano-vesical Centre. A man, aged 30 (*Archiv f. Psych.* Bd. xv. p. 607), fell from his horse, and fractured the first lumbar vertebra. Paralysis of the lower limbs, retention of urine, and loss of power over the sphincter ani at once appeared. The paralysis of the lower limbs gradually diminished, and in less than a year had disappeared. The retention of urine gave way in three weeks to incontinence, which, with the paresis of the sphincter ani, lasted until death. The patient died of pyelonephritis, a year and a half after the accident. The *post-mortem* showed lesion of the conus terminalis at the place of origin of the third and fourth sacral nerves. This is the situation of the ano-vesical centre in man, and it corresponds with the position of Stilling's sacral nucleus.

Zacher on Spastic Symptoms in General Paralysis.—ZACHER (*Archiv f. Psych.* Bd. xv. p. 359) concludes his paper on General Paralysis, by some observations on the spastic symptoms that sometimes occur during the course of that disease.

He finds that the spastic symptoms of general paralysis differ in important respects from the spastic spinal paralysis of Erb and Charcot. In the initial stage of the former there is a peculiar awkwardness and slowness in all the movements, that seems due to a mental want, to the absence of the proper ideas of the required muscular movements, rather than to the muscular stiffness and tension, which operate in spastic spinal disease. Then, again, there are remarkable variations from time to time in the state of the tendon-reflexes, the muscular tension, and the muscular contractures, which are in striking contrast with the regular progression and steady advance of the symptoms in spastic spinal paralysis. In general paralysis the contractures occur almost contemporaneously in lower and upper extremities, and are in both instances flexures; whereas in spastic spinal paralysis, the lower limbs are generally first affected, and exhibit not flexures, but, if we may be allowed the term, extensures (*Streckcontracturen*).

What is the pathology of these spastic symptoms? In two of the cases there was a primary systematic affection of the pyramidal strands quite independent of the cerebral disease; in another case there was a primary non-systematic affection of the cord, which implicated the pyramidal strands, but was found elsewhere and partook of the nature of a multiple sclerosis. Are the spastic symptoms due to lesion of these spinal pyramidal strands, and to lesion of these alone? Zacher answers in the negative. For, there may be spastic symptoms in general paralysis without any disease of the cord; and in the cases reported in this paper there was seen to be a close correspondence between the spastic symptoms and certain brain symptoms, e.g. the paralytic attacks that occurred periodically, and which were probably due to affection of the motor zone of the cerebrum. Zacher concludes that the spastic spinal symptoms of general paralysis may be caused by pathological changes, either in the motor region of the cerebrum, or in the pyramidal strands of the cord, and he thinks that the peculiar course of the symptoms in general paralysis points to lesion of the cerebrum as the predominant factor in these cases.

Westphal states that spastic contractures and rigidity of the muscles never appear in the lower limbs, in cases of combined fascicular disease of the posterior and lateral columns, if the lesion implicates the root-zones of the lumbar portion of the cord. Zacher extends this rule, and enunciates it thus:—In combined disease of the pyramidal strands and posterior columns, spastic

symptoms will be absent in those parts of the body where the corresponding root-zones of the posterior columns are diseased.

The question arises, How is this prohibitive action of the posterior columns to be explained? It is plainly not in harmony with the theory, that spastic symptoms are due simply to direct irritation of the motor fibres of the pyramidal strands. Zacher thinks that the spastic symptoms must be attributed to a pathological exaggeration of certain normal reflex actions, which normal reflex actions may be exaggerated or diminished by influences acting through the pyramidal strands, either in their spinal or encephalic course.

Thomsen and Oppenheim on Sensory Anæsthesia.—The authors contribute a long article (*Archiv f. Psych.* Bd. xv. p. 559 and p. 633) on the occurrence of sensory anæsthesia in diseases of the central nervous system. The term is used to embrace anæsthesias of the special senses, and hemianæsthesias. The paper is illustrated by fifty-two cases, and can only be very briefly summarised here.

The sensory anæsthesias form a typical group of symptoms, the most constant member of which is the bilateral concentric limitation of the visual field; the remaining special senses and the skin and mucous membranes are frequently affected, but by no means so regularly as the visual field. True hemianæsthesias are rare; indeed an absolute hemianæsthesia, in the sense that the other side of the body is quite unaffected, is unknown. Sensory anæsthesia is either a permanent condition, and then there are generally striking variations in its intensity and area of distribution from time to time; or more rarely it is a transient condition. It is important to observe that in almost every case, psychical symptoms are present; there may be disturbances of consciousness—hallucinations, dreamlike states, &c.; or disorders of the affective faculties—irritability, depression of spirits, feelings of apprehension, &c. The degree of anæsthesia runs parallel, as a rule, with the degree of psychical impairment.

Sensory anæsthesia (including hemianæsthesia) is not pathognomonic of hysteria and hystero-epilepsy, as many are apt to suppose. It is found in epilepsy, alcoholism, neurasthenia, chorea; in certain states characterised by continuous nervous apprehension with restlessness; in cases of railway-spine and head-injuries, in multiple sclerosis, in organic brain-diseases, and as a sequela in various psychoses. The authors criticise Charcot's description of

hysterical hemianæsthesia, and dissent from several of his conclusions. They aver that sensory anæsthesia occurs in three forms: first, in conjunction with certain psychical symptoms, just mentioned, as a separate disease; second, in association with other functional neuroses and psychoses; and third, in association with gross organic lesion of the central nervous system. In this last case, it is not the result of any specially localised lesion, but of some general cerebral disturbance. The symptoms are accordingly of little value, as yet, in diagnosis or prognosis.

Bernhardt on Nerve-stretching in Facial Spasm.—BERNHARDT (*Archiv f. Psych.* Bd. xv. p. 777) reports the case of a patient who, after suffering four-and-a-half years from right-sided facial spasm, underwent the operation of nerve-stretching. The spasm disappeared, but a corresponding facial paralysis took its place. Four months afterwards the involuntary convulsive twitching reappeared. Bernhardt gives an analysis of the results of nerve-stretching in seventeen cases of facial spasm, and he comes to the conclusion, that the operation procures temporary relief only; but that it is worth performing, even to secure this temporary respite.

W. J. DODDS, M.D., D.Sc.

On Spinal Sclerosis of Vascular Origin. DEMANGE (*Revue de Médecine*, October 10, 1884).—Female, age 75. No history of syphilis. Pain in loins and lower abdomen; frequent micturition and catarrh of bladder. Later, pain in legs, spasmodic flexions of legs. Later still, permanent contractures, flexion of knees, adduction of thighs; some atrophy; absence of tendon-reflexes; spasmodic action of bladder. Slight systolic cardiac murmur. Finally, sores on the heels, rigidity of neck and muscles of back; death from bedsores.

Post-mortem.—In the heart, granulo-fatty myocarditis, and slight chronic endocarditis; atheroma of the coronary arteries. Atheroma of the aorta and the cerebral vessels.

The spinal changes are characterised by the author as interstitial myelitis, in distribution diffuse and perivascular. There was *posterior sclerosis* both in the columns of Goll and of Burdach, but sometimes of each separately, sometimes together: the sclerosis was not bilaterally symmetrical; sometimes the whole column was involved, sometimes it was entirely sound.

The *antero-lateral columns* were deeply affected, but the crossed pyramidal tracts were not accurately marked out; the direct

pyramidal tracts were for the most part free: the sclerosis extended round the cord to the anterior root-zones.

The *grey matter*: there was one patch of softening in the lower dorsal region; elsewhere it was intact, the anterior cornual cells were normal.

As to the minute anatomy—in the small vessels the endothelium was proliferating and thickened, the vascular walls were thickened and infiltrated with nuclei; the lymphatic sheaths of the vessels were filled with leucocytes. The large vessels were affected in a similar way. The sclerosis appeared to start from the neighbourhood of the vessels: in patches of a star-shaped form with ill-defined borders. The neuroglia was thickened; there were numerous spider-cells; the myeline sheaths were atrophied; the axis-cylinders were present, but not swollen.

The author considers the case to be neither combined systematic sclerosis, nor lateral amyotrophic sclerosis, nor disseminated sclerosis; but diffuse myelitis depending on general atheroma of the vessels.

Acute painful Paraplegia. DUMOLARD (de Vizille) (*Revue de Médecine*, July 1884, p. 532).—An affection observed by the author at Vizille. Six cases are detailed, and the general summary of the symptoms is as follows: The disease begins with lumbar pain and stiffness in the back; the pain may be acute from the onset, or may gradually increase in intensity. It sometimes passes up to the neck. Next, pain in the thighs and legs, not following the course of the sciatic nor limited to the joints, but involving the whole limb. The gait is limping. In severe cases there is slight febrile action, but as a rule the general health remains good, except for pain and sleeplessness.

In a few days the patient is confined to bed with the pain, which is very severe. The reflex function of the cord is much increased, so that the least communicated movement gives rise to a species of tetanus in the lower limbs. The patient is unable to use his legs and has difficulty in passing urine, though the author does not think there is any true paralysis. In rare cases the symptoms are abruptly transferred from the lower to the upper extremity.

The acute period lasts 10 or 15 days; the pain first disappears, then control over the limbs and bladder is re-established. In 30 or 40 days the patient is well.

The pains are not of a shooting character, but rather of the nature of muscular cramp and twitching. They are more severe

than the pains of myelitis, and there is no girdle pain. The cause is as yet unknown. The author compares the disease to an epidemic observed fifteen years ago, at Azannon, in Spain.

On Tabetic Entorrhœa. G. H. ROGER (*Revue de Médecine*, July 1884, p. 555).—The occurrence of diarrhœa in tabes has been commented upon by various authors, *e.g.* Charcot, Vulpian, Fournier, Pitres, Putnam. Out of 32 tabetic patients examined by the author, 14 had diarrhœa: and in 5 of these cases there was reason to think it had special connection with the disease.

“Tabetic diarrhœa” may occur either as an accompaniment of abdominal pains (crises entéralgiques); or without pain (crises interrhoïques), as a mere intestinal flux. Generally it is an early symptom, sometimes occurring even before the lightning-pains, sometimes it dates from the same time as they do, and it may have a distinct relation to them.

The attacks of diarrhœa are sudden both in onset and disappearance, and no cause can be assigned for them. They may last from two or three days up to a week or more. The stools are from three to eight in number per diem, a watery or mucous liquid, in colour grey, greenish, or yellowish. The patient may be subject to these attacks for many years. Not unfrequently they are associated with gastric crises, sometimes with secretory disturbances, such as sweating, sialorrhœa, or polyuria. In a case given by the author the diarrhœa was associated with excessive flow of saliva, and both were checked by the administration of atropine.

Left Inferior Facial Paralysis. Left Hemiplegic Epilepsy. Doubtful Diagnosis before and after Death. LANDOUZY and SIREDAY (*Revue de Médecine*, December 1884, p. 984).—An unmarried female, age 29, whose personal and family history was unexceptionable, came to the Charité for the following complaints: left facial paralysis, involving the lower facial district only, of six or seven months' duration; attacks of convulsions involving the left side of the face, which had begun about the same time as the paralysis as mere twitchings, but had increased in intensity, and at the time of the observation amounted to epileptiform attacks, spreading to the left side of the neck and to the left arm. There was evidence of a slight mitral stenosis, but no other disease.

An attack soon after admission is described as follows:—Slight aura = sense of weight in frontal region, dizziness, tremor of facial

muscles; then sudden attack, stiffness and convulsive movements of left arm, rotation of head and eyes to left, convulsions of facial muscles, tongue protruded and torn by teeth; only slight tremor of left leg. The convulsions were purely unilateral. Duration half a minute to a minute. Consciousness not lost. Paralysis of the affected muscles, and chiefly of the face followed the attack. Maximum interval two or three hours; attacks sometimes subintraant.

The patient became worse, the attacks more frequent and followed by coma, the pupils unequal, the temperature raised. Vigorous treatment with iodide of potassium was instituted, but the patient died.

No lesion was found post-mortem sufficient to account for the symptoms. It is mentioned, however, that on the posterior part of the 2nd frontal convolution, and the corresponding part of the 3rd frontal (right side), there was a patch the size of a five-franc piece, of a pale pink colour. There was a similar appearance on the lower part of the right parietal lobule at the level of the pli courbe.

These appearances, the author thinks, were more likely the result than the cause of the convulsions.

The diagnosis made during life was "Jacksonian epilepsy," i.e. epilepsy symptomatic of an irritative lesion of a part of the right fronto-parietal lobe. The absence of an appreciable organic change post-mortem made this diagnosis doubtful; but there are also difficulties in the way of the other hypotheses which might be advanced, viz. (1) simple epilepsy, with convulsions of limited area, and without loss of consciousness; (2) hysterical attacks having a fatal issue.

Case of Arsenical Paralysis. By CHARLES K. MILLS, M.D. (*Journal of Nervous and Mental Disease*, January 1883).—The poisoning was produced by eating a pumpkin pie containing arsenious acid. The patient was a man aged 24, previously healthy. Vomiting and prostration were the only immediate symptoms. Six days after the poisoning, numbness and aching about the knees came on, followed by weakness in the legs. Three days later, the same phenomena in the wrists and fingers; then complete paralysis below the knees. A month after the poisoning, severe pain, beginning in the knees and spreading downwards, aching, boring, and lancinating in character; the hands being similarly but less severely affected; numbness of the legs also became extreme. The result of an examination two and a half months after the poisoning

was as follows:—Much wasting of the limbs. Legs quite paralysed below the knees: no definite contractures, but occasional cramping pains with involuntary flexion at the knees. Impairment of tactile sense, with extreme sensitiveness of muscles, up to the middle of the thighs. Farado-contraction of muscles absent in legs, diminished in thighs. With galvanism, contraction to medium currents, alteration of polar reactions. Upper limbs:—well marked, though incomplete paralysis below the elbows, affecting specially the supinators and extensors. Movements of thumbs and fingers impaired; well-marked contractures at elbows; Farado-contraction diminished; to galvanism, reaction of degeneration, though less marked than in legs. Hyperæsthesia of fingers. Transverse white bands were noted on the fingers.

Patellar-tendon-reflexes absent. Cremasteric reflexes vigorous, and presenting some abnormalities.

Bowels torpid, some dribbling of urine. No albuminuria. Pulse between 107 and 148. Temperature normal or nearly. The patient improved under treatment, and in three months' time seemed on the road to recovery.

Of six other persons who partook of the pie, four had symptoms of paralysis.

McNutt on Double Infantile Spastic Hemiplegia (*American Journal of Medical Science*, Jan. 1885).—Dr. S. J. McNutt reports a case of double infantile spastic hemiplegia, with notes of the post-mortem appearances, illustrated with seven cuts exhibiting the lesions found. This is believed to be the third, or, at the most, the fourth case of its kind upon record. Yet these cases do not appear to be so very uncommon, since four others presenting similar symptoms are known to be now in New York city. As a distinct condition, even simple infantile spastic hemiplegia has but lately received attention in text-books. For this reason, and on account of the difficulty of obtaining any comprehensive information on the subject, the collection of facts and theories presented in Dr. McNutt's paper is of value, and must lead to a further study of this interesting condition. Dr. McNutt has collected and tabulated 34 cases in which autopsies have been made, and each of them presented atrophy of the cerebral cortex, near the fissure of Rolando.

The subjects of infantile spastic hemiplegia may live to old age. The inception of the disease, however, always dates back to early childhood, or to intra-uterine life. At whatever age seen, its victims are characterised by more or less complete hemiplegic

motor inability, atrophy, and contractures, with or without aphasia, monosyllable utterance, dysphagia, dyspnoea, and idiocy, the latter being especially characteristic of the double affection.

The etiology of infantile spastic hemiplegia has been defined as primitive defect, arrest, encephalitis, and hæmorrhage.

Clinically the cases may be divided into three classes; those in which the inception of the condition precedes birth, those in which it occurs after birth, and those of which parturition is the cause. The paper concludes with a careful study of the differential diagnosis and treatment.

Osler on Jacksonian Epilepsy and the Situation of the Leg Centre (*American Journal of Medical Science*, Jan. 1885).—The author describes a case of Jacksonian epilepsy, the main points of difference between which and true epilepsy are, the slow onset, local in character, beginning in, or in mild attacks confined to, one limb or a single group of muscles; the gradual extension until the side is involved, or, in severe attacks, the entire body; loss of consciousness late, not early and sudden, as in true epilepsy; and, lastly, the muscular contractions are clonic.

His case lasted over fourteen years, the convulsions beginning in the left hand, at first monobrachial, then extending to the leg, afterwards becoming unilateral, and finally general; at first without loss of consciousness. For the first nine years of the illness, there were remarkable intermissions, lasting for six or seven months, once an entire year. Six years after the onset, the left leg got weak and stiff. For four years, the tenth, eleventh, twelfth, and thirteenth of the illness, the seizures were frequent. During this period, there were six weeks of unconsciousness in which the spasms were very frequent, fifty to eighty in the day. Ten months prior to the final attacks, there was freedom from convulsions. The intellectual faculties were unimpaired.

The case is unusual in the limitation of the lesion to the ascending frontal convolution and to its fasciculus of white matter, scarcely involving the grey substance which is commonly affected in cortical epilepsy. The accurate localisation and the remarkable absence of tissue changes in the immediate vicinity give the case the nature of an exact physiological experiment. With this limited lesion of the motor area there were permanent paralysis, with contracture of one extremity, and epileptiform convulsions. Another feature of interest in the case is the light it throws on the situation of the leg centre. The fibrous mass was situated entirely

within the anterior part of the paracentral lobule limited in extent, confined chiefly to the medullary fibres of the superior frontal fasciculus, and only touched the grey matter in places. A point to be referred to is the absence of the paralysis of the leg for the first six years, for, if the convulsions and monoplegia were caused by the same lesion, how explain the late onset of the latter? From the fibroid state of the tumour it might reasonably be inferred that it was originally larger and had shrunk, but the absence of puckering on the surface, and the way in which the margins merged with the contiguous parts, make it probable that the growth was always small; so small, in fact, that at one period of its development it may have caused sufficient irritation to induce the convulsions, and yet at the same time not involve the special fasciculi of white fibres to the extent of producing weakness of the leg, or monoplegia.

Kemper on a Case of Lodgment of a Breech-Pin in the Brain; Recovery (*American Journal of Medical Science*, Jan. 1885).—A lad received a compound fracture of the frontal bone, immediately above the right frontal sinus, by a bursting gun. The breech-pin was found imbedded in the brain, at a distance of half an inch, and was withdrawn by the aid of dressing-forceps. No untoward symptoms were developed until the evening of the fourth day, when a convulsion ensued because of pent-up pus, and after the removal of the cause no further trouble followed. The lesson to be derived from the study of the case is the necessity of maintaining free draining in cases of a similar nature.

Eliot on Poliomyelitis Anterior in Adults (*American Journal of Medical Science*, Jan. 1885).—The author describes a case of poliomyelitis anterior occurring in an adult. The progressive development of muscular weakness, unattended by febrile symptoms, but accompanied by diminution of the size of the limbs, by abolition of the patellar tendon reflex, and by sensations of numbness, yet without loss of tactile sensation, and without interference with the function of either the rectum or bladder, rendered the diagnosis clear and indisputable.

A large proportion of the reports of cases which have been published contain little or no information concerning the details of treatment, and in many others the multiplicity of drugs prescribed renders any reliable conclusions in regard to the effect of each almost impossible. Bromide of potassium, belladonna,

strychnia, ergot, and iodide of potassium have been most often employed, and most praised. Counter-irritation, baths, rubbing and exercise, and electricity are also included as important elements in most plans of treatment. From a careful study of the results of various plans of treatment as reported by various observers, Dr. Eliot deduces the following conclusions:—

First. Counter-irritation and ergot should be employed early in every case. *Second.* Massage and electricity should be used as soon as there is evidence of improvement. *Third.* Little, if any, effect can be expected from iodide of potassium. *Fourth.* Belladonna and the bromides should be used only with extreme caution. *Fifth.* Strychnia should be entirely avoided.

Reichert on the Regeneration of the Vagus and Hypoglossal Nerves (*American Journal of Medical Science*, Jan. 1885).—Since the time of Fontana the subject of the regeneration of cut nerves has been one of great interest and importance, and some experimental work has been done with more or less success. At the present time there seems to be no difference in opinion as to the fact, that fibres of the cut ends of nerves will unite with similar fibres; and that the regenerated sensory nerves will still convey sensory impulses and the regenerated motor nerve motor impulses. In the case, however, of the regeneration of sensory with motor fibres, there yet exists considerable uncertainty.

Dr. E. T. Reichert records some experiments which were made to learn if the fibres of nerves of entirely different origin and function would unite, and if regeneration should occur, to know the form of the return of function, or, in other words, to know if a motor nerve was capable of conveying impulses peculiar to another motor nerve. The vagus and hypoglossal were selected as being nerves of distinct origin and function, and in case of regeneration would probably afford the best facilities for accurate observation.

The experiments were performed on dogs, and it was found that the motor fibres of the vagus in all of the five dogs operated upon had actually become united to similar fibres in the trunk of the hypoglossal, and that the hypoglossal fibres conveyed impulses which were peculiar to the vagus apparatus. Moreover, that in at least one dog (the others not being examined in this way) irritation of the sensory fibres in the hypoglossal trunk gave rise to impulses which were conveyed by the sensory fibres of the vagus to the vagus centres, and produced effects like those induced by

excitation of the vagus trunk, thus showing in both instances that a motor or sensory nerve can convey impulses peculiar to another motor or sensory nerve of entirely different origin and function ; and indicating that at least in some nerves the effects produced by impulses from the periphery are not dependent upon any peculiarity of impulses due to physiological peculiarities of the peripheral sense-organs or nerves through which the impulses are conducted, but upon the peculiar physiological properties of the nerve centres; hence we have respiratory movements, &c., occurring in the tongue brought about by impulses from the vagus centres through the hypoglossal nerve, and effects on the respiration, pulse pressure, and vomiting centre, through impressions carried to the vagus centres by impulses generated in the hypoglossal.

Not only did Dr. Reichert find motor fibres of distinct origin and function united, but we find among the vagus fibres at least three physiologically distinct sets of motor fibres united with fibres of the hypoglossal, viz. fibres conveying *inspiratory* impulses, fibres conveying *expiratory* impulses, and fibres conveying *oesophageal* impulses; the first two sets, no doubt, consisting of fibres of the vagus going through the recurrent laryngeal to the muscles of the larynx, and the latter set forming part at least of the fibres belonging in the same branch.

Another interesting fact to be noted is, that the sensory fibres in the trunk of the hypoglossal, at the point of union with the vagus in these experiments, are recurrent fibres (sensory fibres coming from the superior cervical nerves through the descending branch of the hypoglossal and running from the branch towards the centre), and accordingly conduct impressions normally, not directly toward the centres, as is commonly the case with sensory nerves, but first peripherally making a circuit, as it were, before reaching the centres; therefore, since the sensory fibres in the hypoglossal which united with the sensory fibres in the vagus, conducted impressions to the vagus fibres, it is obvious that these impressions were conducted in a direction opposite to that of the normal, thus offering corroborative testimony to the very interesting experiment of Paul Bert, in showing that sensory fibres can convey impressions in both directions.

Cortical Lesions of the Brain.—By M. ALLEN STARR (*American Journal of Medical Sciences*).—The author gives an analysis of 50 cases of cerebral disease recorded in America, selecting only

those in which there was a single lesion, of moderate extent, of considerable duration, and determined with sufficient accuracy post-mortem. A consideration of the symptoms, both positive and negative, in these cases leads him to the following results.

Lesions of the frontal lobes (excluding the ascending frontal convolutions).—Twenty-three cases of various kinds—wounds, abscesses, tumours. Decided mental disturbance occurred in one-half the cases; a disturbance which may be described as a loss of self-control, and consequent change of character; a loss of power to fix the attention, or to recognise the import of an act in connection with other acts. No symptoms of aphasia, no motor or sensory paralysis; blindness, when it occurred, was referrible to optic neuritis, and disturbance in the sense of smell to pressure on the olfactory bulb.

Lesions of temporal lobes (11 cases).—Such lesions may exist without any localising symptoms; but they may cause disturbances of the special senses of hearing and of smell; of hearing, probably, when the first temporo-sphenoidal convolution is damaged; of smell, when the inner sphenoidal convolutions are damaged. It must be remembered, however, that the cases bearing on the localisation of the sense of smell are few, and that hearing may be impaired by pressure on the auditory nerve. In one case the supra-marginal convolution was affected also, and there was amnesic aphasia.

Lesions of occipital lobes (10 cases).—In these the most prominent symptom was disturbance of vision. (The author has already fully treated of the function of the occipital lobes with respect to sight. *American Journal of Medical Science*, Jan. 1884, abstracted in 'BRAIN,' No. 28.) In three cases there were mental symptoms—loss of memory, occasional maniacal excitement, inability to recognise friends. Lesion of the angular gyrus was found in a case of paraphasia with agraphia; the patient at one time had lost memory of the appearance of words.

Lesions of the parietal lobules (4 cases).—No symptoms could be with certainty connected with the lesion.

The author will, in a future paper, consider lesions of the central convolutions. The present paper indicates, by the method of exclusions, that the centres for motion and for general sensation must lie here.

Multiple Neuritis. By S. G. WEBBER, M.D. (*Archives of Medicine*, August 1884).

CASE I.—Male, æt. 22. Well till a year ago; since then, attacks of darting pains in abdomen and back. For the last five days pain and swelling in legs and arms, headache and feverishness. On admission, tenderness over left radial nerve, biceps cubiti of both sides, and muscles of right forearm; over peroneal and sciatic nerves of both sides, over calves and thighs. Swelling of hands, feet, and face. Arms contracted, in position of partial flexion. Lower limbs more or less paralysed throughout. Plantar reflex most marked on left side; patellar reflex absent on both sides. Faradic contractility feeble in tibialis anticus, peronei, and vastus externus of both sides; absent in rectus femoris and vastus internus; in interossei, moderately good; in extensors of forearms, supinator longus, and biceps almost absent; somewhat better in triceps, deltoid and flexors; in facial muscles normal. Temperature raised for first three weeks (once 104·5°).

He was treated with salicylic acid, iodide of potassium and electricity. Could get about in 2½ months; and in 4 months was quite well. The patellar reflex had, however, returned only in the right leg, and that slightly.

Causes alleged by patient, that he had eaten some unwholesome meat, that he had “caught cold.”

CASE II.—Female, æt. 28. Well till two months ago, when she caught cold while menstruating. After this, pain in abdomen, followed by numbness and anæsthesia, with a (transitory) girdle sensation. Then the numbness left the abdomen, and passed to the knees and legs. Sometimes sharp pains, sometimes dead aching, in the legs. Unable to stand, can move legs and feet, but keeps them slightly flexed; passive extension of them causes disagreeable feeling at heart. Slight numbness of hands. Tenderness over muscles and nerve-trunks of lower limbs. Impaired sensibility in left leg; plantar reflexes good; patellar reflexes absent. Reaction to faradism normal in tibiales antici; poor in right peronei, gastrocnemius, and quadriceps cruris; poor in left gastrocnemius, absent in left rectus femoris. Subsequently the reaction was absent from both peronei and from left vastus internus. Galvanic reactions (both sides) normal in tibialis anticus; in peroneus, “slow but strong” to 12 cells; in gastrocnemius, “very sluggish” to 16 cells.

Pain and tenderness increased in legs, and spread to upper

limbs; subsequently the legs became free; but the fingers anæsthetic and tender.

Treatment: salicylic acid, blisters, morphia, and compresses of carbolic-acid solution.

In five months could walk with assistance, the heels not touching ground. In six months she was well.

CASE III.—Female, æt. 34. History of rheumatism and alcoholism. Attack of vomiting three months before present illness. Present illness three weeks' duration; began with pain and numbness in the legs. Now pain chiefly in joints and back; cannot bear finger ends to be touched; perspirations, delirium. Tenderness of muscles and nerve-trunks of limbs. Paralysis of hands, feet, and legs, and (?) thighs. Anæsthesia in left lower limb. Absence of plantar and patellar reflexes. Complains of choking. Death the day after admission.

CASE IV.—Female, age not given. History of gonorrhœa three years previously, and of chronic alcoholism. Drinking bout four weeks before admission, followed by vomiting, delirium, and hallucinations. For some days had had aching pains in lower limbs, and numbness in hands and forearms. Severe pain on admission; knees flexed, pain on attempt to straighten them; loss of power in extensors of wrists and hands; tenderness over nerve-trunks and muscles of all the limbs. Sore throat, but diphtheria could be excluded.

The paralysis advanced; anæsthesia of fingers and feet appeared; patellar reflexes were absent. The voice became reduced to a whisper. She died seventeen days after admission, with paralysis of the diaphragm, having been too ill to admit of a detailed examination.

Post-mortem.—There were found small suppurating spots in the lungs, and suppuration in the pelvis of the kidneys; also disease of the liver. The brain and spinal cord were both healthy. The principal nerve-trunks of the limbs were found to be diseased; the disease being most marked in the lower end of the sciatic system; less marked, though still extensive, in the distal ends of the nerves of the arms. The changes were very similar to those found in secondary degeneration after section of a nerve, viz. breaking up of the myeline, which was reduced to fine debris, or even absorbed; destruction of the axis cylinders or solution of their continuity; presence of small masses (nuclei?) which stained deeply in carmine. These changes were much more marked at the distal than at the proximal parts of the nerves. Where least marked,

the change consisted only in an alteration of the myeline near Ranvier's constrictions, without increase of nuclei, and without break in the axis cylinder.

The author has observed no less than eighteen cases of this disease, and gives the following summary of his observations:—Ages of patients: minimum, 9 years old; maximum, 51; usually between 20 and 30; no particular preference for either sex; onset a week to four months before admission, recovery usually in a few months; death three times. Sensory disturbances usually prominent; tingling and numbness, then pain and hyperæsthesia, tenderness over muscles and nerve-trunks (especially the latter). Motor disturbances, at first stiffness due to pain, then paralysis; contraction of lower limbs in position of semiflexion is common. Electrical reaction of muscles altered; wasting of muscles and general loss of flesh; sometimes glossy skin, cedema of limbs, excessive sweating. Tendon-reflexes usually absent; skin-reflexes may be normal. Some fever at first; sometimes mild delirium or hysteria. Disease usually begins in legs; may spread to arms, or even respiratory muscles. Diagnosis from anterior poliomyelitis, progressive muscular atrophy, lead-palsy; or in certain cases from rheumatic fever or spinal meningitis.

A Case of Acute Poliomyelitis in the Adult. By JOHN VAN DUYN, M.D. **A unique Case of Poliomyelitis Anterior Acuta of the Adult.** By AMBROSE L. RANNEY, M.D. (*Archives of Medicine*, August, 1884.)—In both cases the authors seek to establish, that excessive muscular action of a limb has an influence in determining the part on which the disease chiefly falls. In Van Duyn's case, the patient had been engaged a fortnight previously in "putting the stone," using principally the right arm. The motor paralysis (which was accompanied by a soreness and tenderness extending gradually over the whole body) began in the right hand, and spread in three days' time to all four limbs, sparing, however, the left forearm and hand; and when seen by the author about four months after the onset, the right upper limb, the left shoulder-muscles, and the left tibialis anticus were principally affected. In four months more, the left shoulder and tibialis anticus had recovered, while there was still much atrophy in the right upper limb.

In Ranney's case, the patient, a sawyer, was accustomed, in the course of his work, to work a treadle with his left foot, and push a board against a circular saw with his right hand. The paralysis

(which developed suddenly after sleeping on wet grass) was confined to the left foot and leg and right upper limb. The paralysis of the leg disappeared in a few weeks; but the right hand and arm remained, after nearly two years, paralysed and atrophied.

Illustrations of the Anomalous Course of Posterior Spinal Sclerosis.—By E. C. SEGUIN, M.D. (*Archives de Médecine*, October 1884).—In the first two cases the onset of the disease was marked by symptoms suggesting focal lesion of the lower part of the cord.

CASE I.—Old lateral curvature of spine; syphilis fourteen years ago. Two years before he first came under observation, numbness appeared in left foot and leg and spread recently to right. Symptoms on first observation: progressive weakness of legs, slow micturition, diminution of sexual power; anaesthesia of feet: loss of patellar and plantar reflexes. No pains, no ataxia. A year later, ataxia had developed; and in another year or two, fulgurating pains, first in rectum, then in thighs and legs.

It is possible that a syphilitic myelitis occurred first, centrally situated, in the lower part of the cord, and posterior sclerosis followed secondarily.

CASE II.—The patient, who had been previously healthy, except for a chancre some years before, came under observation for what appeared to be acute double sciatica, continuous pain in both lower limbs, which had come on suddenly after a chill. Numbness, slight loss of power, and slight emaciation of the legs followed; there was paralysis of bladder, absence of tendon-reflexes, well-marked ataxia of legs, griping pain in left chest. All the original symptoms disappeared, excepting the ataxia of the legs; the patient became subject to the ordinary fulgurating pains, and acquired the characteristic gait of locomotor ataxy.

The possibility of a local syphilitic lesion is again pointed out.

CASE III.—Diplopia and atrophy of the optic nerves appeared four years before the pains set in.

CASE IV.—The neuralgic pains had existed for twenty-nine years without ataxia of the legs. The patellar-tendon reflexes were absent. The patient died from cardiac and renal disease: post-mortem, sclerosis of the lateral parts of the posterior columns was found to exist.

CASE V.—Characteristic pains for twelve years; slight anaesthesia of feet, absence of tendon-reflexes: no ataxia. Chronic arthritis of both knee-joints.

CASE VI.—A patient had previously had two attacks of “cerebral congestion,” and had once been “almost insane.” He consulted the author for lightning pains of six years’ duration. Absence of patellar tendon-reflexes, *slight* staggering with eyes closed. In three years’ time, typical general paralysis with grand delusions had developed.

Such a complication is well known to occur in the later stages of tabes: the author thinks it is unusual in the early (præ-ataxic) stage. In the present case, the cerebral and spinal affections may have been independent of each other.

The Neurotic Disturbances after Joint Affections. By Dr. GEORGE W. JACOBY (*Journal of Nervous and Mental Disease*, April 1884).—The paper is based on the observation of thirty cases; three of which are briefly detailed. These nervous disturbances may affect (1) sensation—producing hyperæsthesia or hyperalgesia (usually at an early stage), or anæsthesia (usually later), or neuralgic pains; (2) motion—producing paralysis of the muscles; (3) nutrition—producing muscular atrophy, hypertrophy of the subcutaneous tissue, sclerosis of the skin. The author’s remarks chiefly relate to the muscular atrophy and paralysis. The course of the affection is as follows. At a variable time after an injury to a joint, which may have been severe or slight, the muscles become flabby, or even wasted, and lose their contractility. Paralysis (i.e. loss of volitional contractility) usually precedes this wasting; but on testing the wasted muscle it is found that their electrical contractility is affected also. Contractility both to faradic and galvanic current is much diminished, but there is no reaction of degeneration. This paralysis and atrophy affects, first and principally, the extensor muscles of the injured joint; but it may spread to others; and has been reported to pursue an ascending course and involve the entire half of the body (Révellont). In the case of the ankle- or wrist-joints, however, the extensors are not specially singled out, and the affection has a descending course. In the case of the finger-joints the interossei suffer most. The author believes that in paralysis of this kind there is always muscular atrophy, but that it may be obscured by hypertrophy of the subcutaneous tissue. In one of his cases, after injury to the knee, paralysis of the triceps cruris was noted as soon as three days subsequently, and atrophy in four days more. The diagnosis from progressive muscular atrophy may be in some cases difficult, especially where the interossei are involved; the points of distinction are—that the paralysis precedes

the atrophy, disturbances of sensation are oftener present, the reaction of degeneration is absent. The prognosis is favourable: for treatment, the author relies greatly upon massage.

Case of Nodular Tumour of the Corpus Callosum. By FRANCIS A. MCGUIRE, M.D. (*American Journal of Neurology and Psychiatry*, May, 1884).—This tumour (which appears to have been gummatous in nature) was a firm, irregular body, seated on the anterior edge of the corpus callosum, and covering about $2\frac{1}{2}$ cm. of its upper face, also encroaching slightly on the curve towards the rostrum. Length $3\frac{1}{2}$ cm., greatest diameter 2 cm. It consisted of a main mass continuous with the corpus callosum, with buds sprouting from it. The tissue of the corpus callosum was unsoftened, but the adjacent parts of the gyrus fornicatus on both sides was excavated; and also the cortex in the neighbourhood of the ascending and horizontal parts of the calloso-marginal fissure. There was also much thickening of the ependyma of the fourth ventricle.

The author does not connect the clinical phenomenon of the case with the tumour; the course of the disease had been briefly as follows:—syphilis in 1873, meningitis (?) in 1880, fair recovery: left facial palsy in 1882. Next year, headache, convulsive attacks, partial dementia; finally, other fits, in one of which he died.

Case of Tumour in the Optic Thalamus. By T. A. McBRIDE, M.D. (*American Journal of Neurology and Psychiatry*, May 1884).—This tumour (also probably gummatous), of the size of a walnut, sprang from the optic thalamus at the junction of its posterior and middle thirds, and spread inwards into the ventricle, and through the corpus callosum into the longitudinal sulcus. [A transverse vertical section of the brain, made at the fissure of Rolando, thus shows the corpus callosum destroyed in the middle line, and to the right of the middle line.] The patient (who was deaf, incoherent, and dull) had left hemiplegia; and died with rise of temperature and delirium. He had complained previously of headache, and had been unable to walk, or to button his clothes, &c., so well as formerly.

J. A. ORMEROD, M.D.

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